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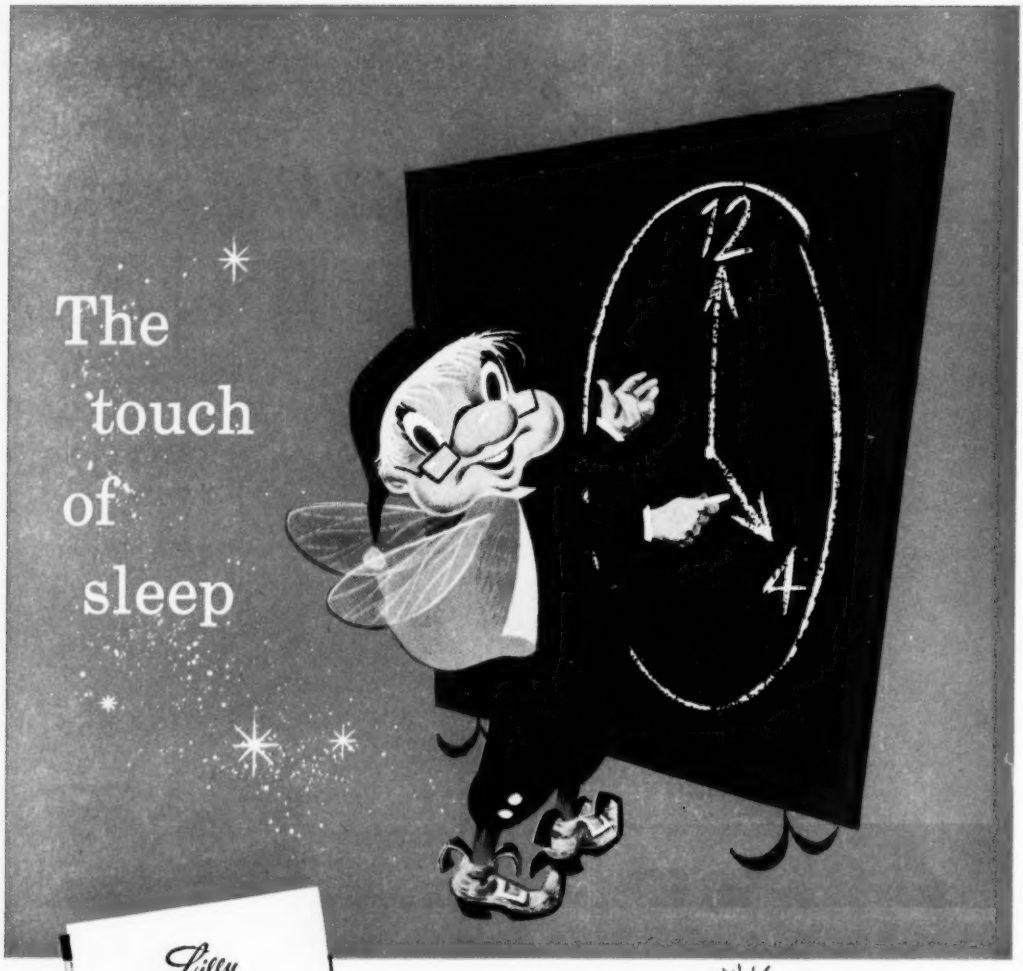

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Southwestern MEDICINE

VOL. XXXVII MARCH, 1956 No. 3

Symposium on Headache*

Introductory Remarks and Differential Diagnosis

By Derek Denny-Brown, M. D., Neurologist-in-Chief, Boston City Hospital; Professor of Neurology, Harvard Medical School

Headache is something that all of us must have experienced at one time or another, and therefore is of general interest. Whether a headache can be attributed directly to our behavior (a "hang-over"), or whether it is a reaction to some stress or disease within us, it is a most distressing symptom. Even in its mildest form it has a high "nuisance value."

As a medical problem headache presents obvious difficulties in investigation. It is a subjective sensation. The investigator cannot measure it directly. In this connection we must acknowledge the important pioneering work of Dr. Wolff of New York, who with his associates has devised most ingenious methods of making comparative measurements of head pain, and of the pulsations of blood vessels of the scalp. In this way, valuable data on headache mechanisms have been collected. Headache, however, has many aspects and in this symposium we are bringing together several different points of view from the more purely clinical aspect of the problem.

SOUTHWESTERN MEDICINE is honored at the opportunity to publish this important and distinguished symposium on "Headache" prepared by the Tufts-New England Medical Center and written by leading specialists in the field. We believe that physicians of the Southwest will find this symposium of the highest interest. — THE EDITORS.

Numerous Kinds

First, however, it should be emphasized that there are a number of different kinds of headache, even of common mild recurrent headaches, and that sometimes, especially when it becomes progressively worse and is associated with vomiting and increasing dimness of vision, headache may indicate very serious disease. But most people are concerned about the kind of headache that comes at intervals in otherwise good health, and may recur for years without any obvious disease. Migraine headache is a common such variety.

Migraine and megrim are very old words derived from the "hemicrania" of the ancients. To many it is known as sick-headache or bilious headache. For the neurologist the "classical migraine" has the greatest interest, for it is the variety in which an aura or premonitory symptom opens the scene. The aura takes the form of an expanding, star-shaped hallucination in the field of vision, or numbness in the mouth or hand, lasting five or ten minutes and pointing to a one-sided disturbance of the parieto-occipital region of the brain. It is always a sensory disturbance. This indicates a very particular liability of the blood vessels of the parietal lobe. These same vessels are those most often affected by anoxia of high altitude type. It is also no coincidence that these blood vessels of the parietal region are reported to be more responsive than other small cerebral vessels to stimulation of the sympathetic nervous system, although sympathectomy does not abolish migraine.

Brain Storm

The brain storm of classical migraine is a self-limited affair that terminates in a few hours, practically invariably ended by sleep. In a susceptible individual it may be set up by any prolonged stress—long hours of work, anxiety or insomnia. But if in this way it is a weak point in a person's makeup, how is it that at another time the same headache may strike him when he is relaxed and enjoying himself? Why does he have this particular liability and how can he abolish it?

Most people are liable to recurrent headaches that are less clear-cut and less dramatic than classical migraine. These appear gradually and without a preliminary warning, may be present on awakening, and may last two or more days. The same throbbing cranial vasodilation that is associated with classical migraine, the same intolerance of rise of venous pressure, occurs with these "migrainous headaches." Their very persistence may lead to more exhaustion and prostration than the stress that caused them. Prostration becomes the leading symptom, and is clearly traceable to mental anxiety. Such headaches pose difficult problems for the physician because the factors originally leading to the bouts of the headache become inextricably confused with anxiety about the headache itself and the inconvenience it has caused. These are often called tension headaches and will be discussed by Dr. John Graham.

*This symposium published by permission of the Tufts-New England Medical Center Bulletin.

Nervous Stress

In other patients nervous stress or anxiety are linked with more general discomfort in the head, such as pressure or bursting sensations. Such sensations commonly accompany nervous depression. How does this happen? Headache cannot be produced by sheer effort of imagination. Some have suggested that the muscles of the neck or the eyes are too tense, but that is surely not the whole explanation, for the pain and tenderness are referred directly to the scalp and even a hairbrush causes discomfort. In traumatic headache, although the tender area is usually an old scar in the scalp, it is remarkable how many factors such as an injection of histamine or simple anxiety can precipitate a characteristic headache. How is the scalp sensitized?

Studies on the effect of various pharmacologic substances on the blood flow in the scalp and brain have given a great deal of information. Dr. Arnold Friedman, a former associate of mine, has made statistical studies of the effects of all the modern remedies on a great variety of types of head pain. He will discuss the use of various substances in treatment on the basis of his experience at the Headache Clinic, Montefiore Hospital, New York.

If headache is always felt in one particular place in the head, it obviously is likely that some abnormality underlies that place. It may not be serious (perhaps just an old scar) or it may be caused by trouble in a sinus or air passage. It is seldom caused by pressure on the brain but may be the result of overdistention of some blood vessel within the skull, or leakage from an aneurysm.

Circle of Willis

The blood vessels of the brain form a network, the circle of Willis. In 1664, Dr. Willis noted that one of his patients had had a complete occlusion of the right carotid artery for many years, and had been tormented by severe left sided headaches. In his quaint way Dr. Willis said "the cause whereof I know not how better to explain, than that the blood excluded from the right Carotid Artery, when at first it rushed more impetuously in the left, had distended the membrane." We now know that this is true, and that certainly in many elderly people, when some arteries are narrowed, the others suffer from painful dilation. This pain at the back of the head is one of the warning signs of insufficiency of the basilar artery; it is as important in neurology as angina is in cardiology. The associated attacks of dizziness with dysarthria and other signs of impaired function in the brain stem are the clue to this kind of disorder. Likewise, distention of the internal carotid artery in its narrow canal, whether caused by head injury or by arteriosclerosis, is associated with a Horner's syndrome with narrow pupil on the side of the headache and distended artery. Some types of brain tumors take an enormous blood supply, draining their area of the circle of Willis. Dr. Bertram Selverstone will discuss the surgical aspects of head pain and its relation to the larger arteries.

Final Discussor

Dr. Bayard Horton, physician to the Mayo Clinic, who is well known for his original description of two very distinctive types of head pain — "histaminic cephalgia" (Horton's cephalgia), and also the syndrome of temporal arteritis, will be the final discussor. Histaminic cephalgia is an unusual kind of headache, so distinctive that once encountered, it will never be forgotten. Its most interesting feature is its brevity. The clear-cut cycle of an attack, its extremely localized and obvious manifestations, all offer more opportunity for direct analysis of the disorder. Yet it still remains an intriguing problem. Transitions between this disorder and classical migraine suggest that the solution of the one will solve the other. Temporal arteritis is also associated with very severe head pain, and Dr. Horton will discuss this and "histaminic cephalgia."

Although the discussion will cover these many varieties of headache, it must not be forgotten that there are still other varieties, especially those that arise from troubles that are primarily unrelated to the head in any way. For example, headache is a common symptom at the beginning of any fever, and is associated with kidney disease and high blood pressure, or with excessive loss of body fluids. What will be discussed chiefly are conditions in which headache is the leading or only trouble, assuming that the presence of other more general disease or disorder has been eliminated by a preliminary examination.

Fourth International Congress On Diseases of the Chest

The American College of Chest Physicians, an international society, has announced that its Fourth International Congress on Diseases of the Chest, sponsored by the Council on International Affairs of the College, will be held in Cologne, Germany, August 19 through 23.

Bone and Joint Surgeons to Meet in Minneapolis

The Association of Bone and Joint Surgeons will hold its annual meeting in Minneapolis March 22-24.

Dr. Louis W. Breck of El Paso is president of the Association.

HALLUX VALGUS

Booth, W. G., Letters to the Editor, Lancet 1:396, 1953

Terminal-phalanx hallux valgus is congenital. Developmental hallux valgus is unknown among children who have always gone barefoot. In England more than 50 per cent of school children have developmental hallux valgus and Booth contends that some all-embracing factor, such as tight shoes, must be responsible. Since hallux valgus disappears in children when properly fitted shoes are worn, it is difficult to accept the statement that there is no known cause. "Every child needs a new shoe fitting approximately every three months . . . How sad it is that this preventable condition is not prevented."

Clinical Clippings, May, 1953.

Mechanisms of Vascular Headache

By John R. Graham, M. D., Chief of Medical Service, Faulkner Hospital; Clinical Associate in Medicine, Massachusetts General Hospital, Boston

The title of this symposium is "Vascular Headache." My particular task is to discuss its pathogenesis. I will confine my remarks to the kind of headache known as "migraine" or "sick headache."

It is of value to consider what has been learned about this type of vascular headache by observation of its attributes and natural history. It is a widespread disease that occurs in about 10 per cent of the population. It is more prevalent in women than in men. Of great importance is the fact that it is familial. It can start to manifest itself as early in life as the victim can make it known, and probably earlier. This must be remembered when one is tempted to attribute its etiology entirely to factors that occur only later in life, such as the presence or disappearance of the menstrual cycle. It is at first, and in most cases continues to be, a periodic disorder that leaves no permanent changes so far detected in its wake. It can disappear entirely for long periods of time only to recur later, or it can gradually become a practically continuous condition as in "migraine status," or it can disappear in middle age never to return.

Perfectionistic Personality

It is more common and apparently more severe in persons with a perfectionistic personality, a delicate body build and high intelligence, but it is not limited to people with these attributes, and there are many people possessing them who do not have vascular headaches. It is a disorder that manifests itself powerfully in a focal area of the body but is also, at times, preceded by, and frequently accompanied by, manifestations of a more general systemic disturbance. There is a tendency in some subjects to alternate typical attacks of vascular headache with episodic disturbances affecting other parts of the body, such as paroxysmal tachycardia, abdominal pain, and vertigo. Although the statistics are not extensive or conclusive, carsickness, allergies, epilepsy and even gout seem to be unusually prevalent among victims of vascular headache and their families.

Headache becomes more frequent as life's stresses increase and diminishes as they grow less (fig. 1).

Headaches recede under the influence of severe illness and surgical operations (fig. 2), an observation that has been confirmed for a variety of operations and illnesses, including jaundice, without any specificity.

Pregnancy has a dramatically beneficial effect on vascular headache (fig. 3).

Since this type of response to operation and illness and pregnancy follows the behavior of rheumatoid arthritis as pointed out by Hench, a trial of cortisone

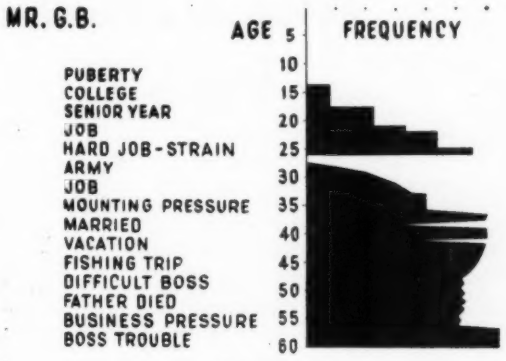
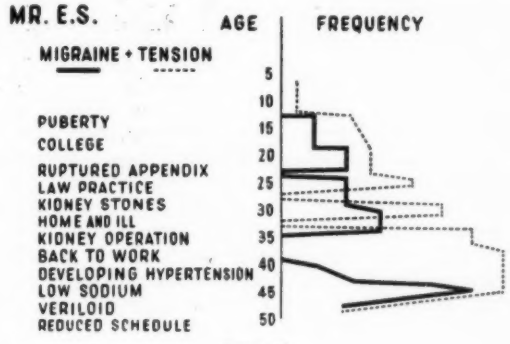


FIGURE 1.



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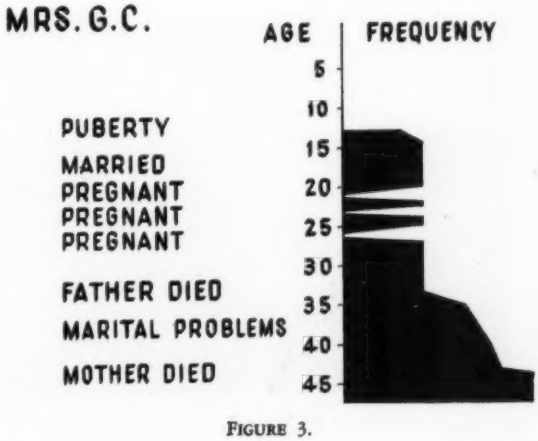

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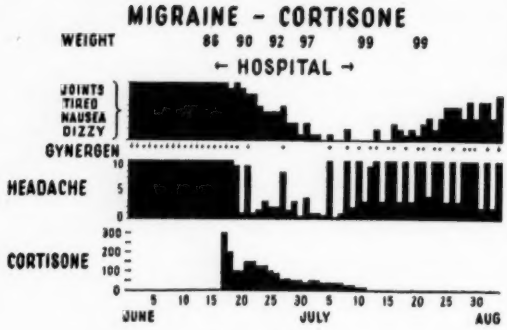

Figure 4.

seemed justified (fig. 4). Relief to migraine while cortisone is being administered was striking; all symptoms returned when cortisone was stopped.

The foregoing are observations of rather major points of behavior. They suggest that vascular headaches are made worse by increased exposure to stress of various types and are relieved when stress is removed, or the patient is removed from stress, or the patient is fortified against stress by the acquisition of additional "stress hormones" either through surgery, illness, pregnancy or the administration of steroids.

Some of the factors which precipitate attacks according to the patients are worth noting: bright light, noise, strong smells, changes in the weather, allergy to certain foods, hunger, physical fatigue, menstrual periods, menopause, emotional upsets and strain arising from arguments, resentments, difficult situations, confusion, crowds, shopping, guests, long drives, crowded schedules, exciting events. To be sure, the last on the list are the most commonly blamed, but the others, having a more somatic character, definitely need to be included.

Of especial interest is the fact that these headaches most commonly start in sleep or early in the morning, are especially liable to occur if the patient sleeps late, and have a sinful ability to spoil Saturdays, Sundays and holidays. They have a definite tendency to occur after the stressful period rather than during or before it. In my opinion they behave as if they were the result or aftermath of an experience rather than a psychological escape from an unpleasant situation that is about to take place.

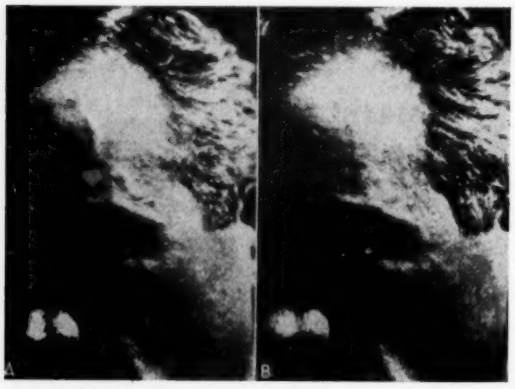

Figure 5.

All of these characteristics of the "sick headache" need to be kept in mind as we approach a more detailed study of the mechanisms involved in the actual attack.

In classical migraine, the attack is ushered in by visual scotomata, aphasia, hemianesthesia, and other striking neurological symptoms. When these clear up, the headache begins. There is considerable evidence that the first phase of this syndrome is associated with constriction of cerebral vessels and cortical ischemia. Pointing strongly in this direction is the fact that during this stage of the attack the administration of cerebral vasodilators such as CO_2 or small doses of nitroglycerin, alleviates the neurological disturbances. Electroencephalographic tracings made during this phase are consistent with cerebral ischemia.

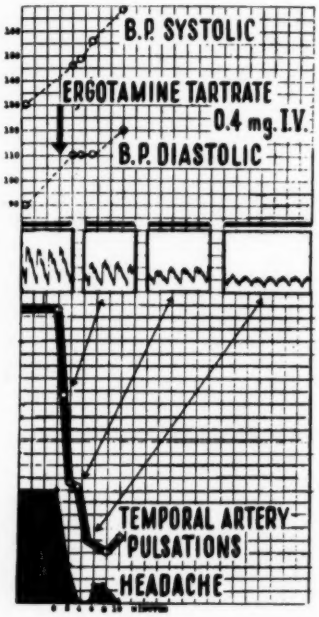

Figure 6.

Even in the "ordinary sick headache" in which the head pain is not preceded by bizarre neurological fireworks, there is evidence that vasoconstriction takes places in more superficial branches of the cranial vasculature before the headache begins.

On the other hand, when one considers the painful phase of the migraine attack, there is abundant evidence to show that the head*ache* is associated with dilatation and excessive pulsation of the cranial arterial tree (figs. 5, 6, 7).

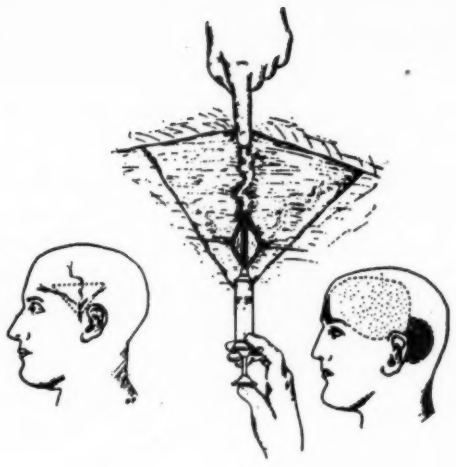

Figure 7

Figure 5 reveals a man on the left suffering from a left temporal headache. There is distention of the temporal vessels. Half an hour later, after the injection of ergotamine tartrate, his head pain had disappeared and the photograph on the right demonstrates the decrease in the prominence of the previously distended vessels.

Figure 6 shows pulse-wave-tracings from this same artery at the time of another headache and during its relief by ergotamine tartrate. Decrease in headache goes hand in hand with decrease in amplitude of temporal artery pulsation.

Figure 7 reveals the same artery at operation. When it was distended forcibly with saline, headache was reproduced; and when the artery and its perivascular coats were ligated and sectioned, the pain of subsequent headaches was relieved in the area supplied by this artery.

Manual compression of the carotid pulse on the affected side relieves headache. Drugs such as pituitrin, nor-epinephrine, ephedrine, and benzedrine, all relieve headache so long as they exert a constricting influence on the cranial vessels involved in the attack. On the other hand, vasodilating drugs, such as histamine and amylnitrite, make headache worse if given during the attack. Thus, the painful phase of migraine is at least associated with cranial vasodilatation and decreases with the cranial vasoconstriction brought about by either mechanical or pharmacologic agents.

This is not proof, however, that the dilatation in itself causes the pain. It is common experience that cranial vascular dilatation such as that occurring in sunburn or exposure to heat does not necessarily cause headache. Consequently, it is postulated that some additional factor, as yet unknown, must be at work at the site of the headache, which affects vessel walls and perivascular tissues in such a way that they respond with pain to vascular pulsation and dilatation.

What is this factor? Why should this vasodilatation be so prolonged and painful? Is the misbehavior of the cranial vessels but a part of a more complicated and deep-seated abnormality? These are the questions that need to be answered by future investigations. The observations made so far present me with the following hypothesis:

The patient with this type of headache has an inherited constitutional diathesis that predisposes him to this disease. Whether this has to do with localizing the condition to the head or has to do with more general systemic disturbances, or both, is hard to say. There is much to suggest that the vascular headache patient belongs to a broad group of patients subject to various relapsing disorders such as rheumatoid arthritis, asthma, ulcerative colitis, gout, and so on, in whom we find a similar relationship of the symptoms to stressful life situations. In all of these we observe an abnormality of response on the part of the adaptive mechanisms of the individual.

Vascular Headache

In the case of vascular headache, the cranial vasculature seems to be the system that is most affected, and there is a strong suggestion that these patients are particularly vulnerable to stresses that have their greatest impact upon the central nervous system. When stress of this type is experienced, the bodily defenses against it react imperfectly, too little or too late or inappropriately so that damage is allowed to occur and headache results. Recent studies reveal the necessity for intactness of the hypothalamic-pituitary-adrenocortical system if damage is to be prevented after strong autonomic nervous system activity, especially as applied to blood vessels. In the vascular headache, the suggestion is strong that the initial vasoconstriction is the result of prolonged or excessive sympathetic activity. The ischemia so produced might well not be so damaging to vessel walls if the supporting adrenocortical system operated quickly and adequately. If the supporting system is inadequate, damage to the vessel wall may be allowed to take place. The damaged vessel dilates and its wall becomes edematous. In this state, it is a source of pain when stretched by excessive arterial pulsation. The severity of the pain and other physical manifestations of the headache finally arouse the sleepy defense system which then goes into action, and brings about a reversal of the pathological process. Patients who inherit or are made with such a deficient defense system naturally get into more trouble if their personality is such that it drives them unduly into fatiguing, stressful situations.

This is only my hypothesis. Other observers are undoubtedly formulating their own, and I am sure there will be many variations and subsequent changes.

Temporal Arteritis

By Bayard T. Horton, M.D., Section of Medicine, Mayo Clinic, Rochester, Minnesota

I have been asked to comment on the headache of temporal arteritis, for it should be included in the "Symposium on Vascular Head Pain."

First described by Horton, Magath and Brown in 1932,[1] and in later publications,[2–5] temporal arteritis was officially recognized as a new disease entity in an editorial in the *Journal of the American Medical Association,* June 22, 1946, based on reports of 38 cases from the world's literature. From 1931 through 1954 a total of 103 patients were observed at the Mayo Clinic.[6] The disease occurs exclusively in older persons, the average age being 69.2 years. Of the 103 patients, 47 were men and 56 were women. The cause is unknown.

All patients with temporal arteritis have headache. It has been a prominent feature in each case. The pain is vascular in origin. The early stages of the illness usually give no clue as to its nature. Severe headache, which is present throughout the waking hours, difficulty in chewing, general malaise and lassitude, night sweats, symptoms of sepsis, anemia and a high sedimentation rate may persist for weeks before the appearance of the typically swollen temporal arteries. This is the foremost clinical feature of the disease. Hence, the term "temporal arteritis" is an appropriate descriptive name for the disease entity. In our series of cases the involved arteries have been resected in 61 per cent of our patients, and "giant cell arteritis" is the dominant histologic feature.

Ocular Involvement

Some type of ocular involvement, ranging from diplopia to complete blindness, has been observed in 53 per cent of cases at the clinic. Of the 103 patients in our series, 21 had bilateral loss of vision, either total or industrial, and 11 had unilateral loss of vision, either total or industrial. Sudden loss of vision in an elderly patient with headache and symptoms of sepsis should alert the physician to the possibility that the patient may have temporal arteritis.

The disease usually is not fatal and tends to run a self-limited course. Some patients have lived as long as 15 and 17 years after recovery from this illness. It is my impression that the disease frequently goes unrecognized.

Cortisone has been used in the treatment of temporal arteritis at the clinic since November, 1949.[7] The incidence of blindness has been definitely decreased since cortisone and corticotropin (ACTH) have been employed in its treatment. The use of cortisone and corticotropin and the intravenous administration of histamine also have been helpful in the symptomatic relief of headache. Removal of large segments of the involved temporal artery has given great relief of headache to many patients.

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Histaminic Cephalgia: Differential Diagnosis and Treatment*

Bayard T. Horton, M.D., Section of Medicine, Mayo Clinic and Mayo Foundation,† Rochester, Minnesota

The clinical picture of histaminic cephalgia must be accurately defined if valid conclusions are to be drawn from the observations that I wish to report. In so doing, I am attempting to describe only what I have seen and what my patients have told me. From 1937 to January, 1955, I have seen 1,176 patients with histaminic cephalgia. Of the 1,176 patients 1,023 were men and 153 women.

Histaminic cephalgia is a distinct clinical entity.[1–9] It occurs chiefly in males and is characterized by a unilateral headache of short duration, generally lasting less than an hour; it commences and often terminates suddenly and tends to awaken the patient at night, one to two hours after he has gone to sleep.

Patients usually refer to their attacks as "head pain" rather than headache. Pain is the outstanding complaint. It is usually steady, excruciating, burning and boring in character. The pain involves the region of the orbit and temple and may extend to the upper jaw, occasionally the lower jaw and into the occipital area, the neck and the shoulder. In the typical, full-blown case of histaminic cephalgia the height of the pain is associated with sweating (fig. 1, a and b), profuse watering and congestion of the eye, rhinorrhea or stuffiness of the nostril and often swelling of the temporal vessels of the involved side of the head.

Suicide Contemplated

So severe and frequent are the attacks of pain that practically every patient has contemplated suicide. I

*Part of the Symposium on Vascular Head Pains at the Tufts Univ. School of Med., Boston, Massachusetts, May 5, 1955.

†The Mayo Foundation, Rochester, Minnesota, is a part of the Graduate School of the University of Minnesota.

have never known one to carry out his threat, probably because of the short duration of the attack and the fact that he feels normal between attacks.

There are no trigger areas and the pain is not confined to the distribution of any cranial nerve. Nausea, vomiting and cortical phenomena are usually absent. There is no hereditary factor; hence, the syndrome should not be confused with trigeminal neuralgia or classic migraine.

Figure 1. Appearance of a man 55 years of age during an induced attack of histaminic cephalgia. The attack developed 20 minutes after the subcutaneous administration of 0.35 mg. histamine base. Beads of perspiration are evident above the left eyebrow and in the left orbital area (a), and rhinorrhea from the left nostril is also evident (b). He had been having one to three attacks each 24 hours. The duration was from 15 to 60 minutes. This induced attack persisted for 20 minutes. He had previously had numerous intranasal operations, cocainization of the left sphenopalatine ganglion and deep injections of alcohol into the first and second divisions of the left fifth cranial nerve, without obtaining relief.

Residual soreness may persist in the region involved with pain for a short time after the attack has ceased.

Attacks tend to occur in a series and may appear to do so on a seasonal basis (fig. 2). Remissions and exacerbations occur spontaneously. Horner's syndrome is not uncommon in this type of case. The latter may disappear, after a given series of attacks has ceased, or may remain permanently.

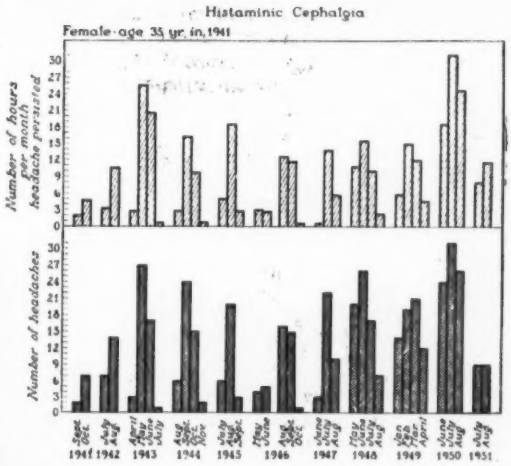
Histaminic Cephalgia
Female-age 35 yr. in 1941
Number of hours per month headache persisted
Number of headaches
1941 1942 1943 1944 1945 1946 1947 1948 1949 1950 1951

Figure 2. A graphic record illustrating the monthly incidence and total duration of attacks of histaminic cephalgia for 11 consecutive years. The patient was a woman who was 35 years of age in 1941.

Night attacks are characteristic of this illness (fig. 3) and often occur with clocklike regularity. The sudden onset of excruciating pain during sleep makes the patient get out of bed — sometimes he jumps out of bed before he is fully awake because assuming the erect posture tends to give slight relief from pain. Many patients have tried sleeping in a chair at night in a vain attempt to avoid such attacks. Some have tried sleeping during the day, but still the attacks have continued to occur during sleep.

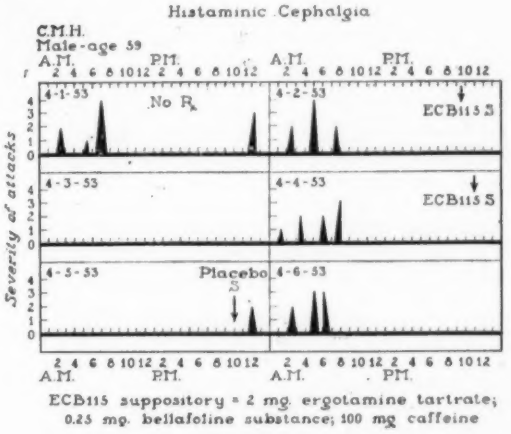

Figure 3.

Graph illustrating number and severity of nightly attacks of histamin cephalgia in a man aged 59 years. He was totally incapacitated and came to the clinic as an emergency case. The use of an ECB-115 rectal suppository at 10 p.m. on April 2, 1953, prevented nightly attacks. This was the first night in twelve previous nights that he had slept without an attack. The suppository

was not used the following night and four attacks developed between the hours of 1 a.m. and 7 a.m. The night of April 4, the ECB-115 suppository was again used at bedtime and the patient slept throughout the night without an attack. Then, on the night of April 5 he was given a placebo suppository which looked exactly like the ECB-115 suppository. He went to sleep but was awakened approximately 1 hour later with an attack, and during the night he had four attacks.

Converting Time of Attacks of Histaminic Cephalgia From Night to Day

Case #1. — A man, aged 34 years, came to the Mayo Clinic on September 3, 1951, because of recurring attacks of histaminic cephalgia, and was admitted as an emergency case. For the previous six weeks he had been having an average of five attacks each 24 hours. All attacks occurred at night, awakening him from sleep. The pain always made him get out of bed and pace the floor. Each attack lasted from 10 to 60 minutes. The pain involved the right temporal and orbital regions and spread down into the right side of the neck. It was very severe. At the height of the pain he invariably noted redness and tearing of the right eye and plugging of the right nostril.

Great Vessels

The great vessels in the right side of the neck were sore when palpated. He was not nauseated and had not vomited. Because of the frequency and severity of the night attacks, it seemed an excellent opportunity to determine whether a rectal suppository containing ergotamine tartrate and caffeine would prevent them. Accordingly, the first day he was at the clinic he was given one ECB-115 suppository to use at bedtime. Each ECB-115 suppository contains 2 mg. ergotamine tartrate, 100 mg. caffeine and 0.25 mg. bellafoline substance. The suppository was used at 10 p. m. and the patient slept the entire night without an attack of histaminic cephalgia developing. For eight consecutive nights at 10 p. m. he used the ECB-115 suppository and did not have a night attack, but he had 22 day attacks during this time.

Day Attacks

As he expressed it, "Doctor, you have converted my night attacks into day attacks." The day attacks began to occur approximately 55 hours after the use of the first ECB-115 suppository. On the ninth day he again went to bed at 10 p.m. but did not use the ECB-115 suppository. He had three attacks during the night. The first attack occurred between midnight and 1 a.m. and was of grade 2 in severity. The second occurred from 1 to 2:30 a.m. and was of grade 3 and the third occurred from 5:40 to 7:30 a.m. and was of grade 4. He had been free from attacks of histaminic cephalgia for four days and nights prior to his dismissal on September 20.

Discussion

Patients with tension headaches and migraine-tension headaches may occasionally be awakened at night with headache, but these attacks lack the explosive character experienced by the patient with the histaminic cephalgia syndrome. The patient having migraine-tension headaches is more apt to remain in bed than to get up and walk around.

In order to contemplate the clinical picture created by headache, one must have a starting point. A carefully recorded history will usually furnish more clues as to the underlying cause of the pain than will all other diagnostic procedures combined.

Since the turn of the century, many new mechanical devices have been brought forth to aid the physician in obtaining more accurate data regarding the patient, but little or no progress has been made from the standpoint of obtaining a more accurately recorded history.

Adequate treatment cannot be instituted until the type of headache has been determined. The patient should have a complete physical and neurologic examination; the eyes, ears, nose and throat should be carefully examined; roentgenographic examination of the head should be employed routinely, and special diagnostic tests also may be indicated in cases in which the presence of organic lesions is suspected.

Clinical Experience

The physician with a rich clinical experience gained from contact with patients should have little difficulty in differentiating histaminic cephalgia from classic migraine, trifacial neuralgia, atypical facial neuralgia and other head and face pains.

Histaminic cephalgia is a medical, not a surgical problem, and yet, approximately 40 per cent of my patients have previously undergone unnecessary operations in an attempt to obtain relief from their pain.

The transmission pain pathways of histaminic cephalgia have not been worked out completely. White and Sweet,[10] in their recent book on pain, have a chapter dealing with this syndrome to which they refer as "autonomic faciocephalalgia (Horton's syndrome)." They differentiate Horton's syndrome from migraine. A chapter entitled "Cephalic Pain—Transmission Pathways Uncertain" concludes with this significant statement, "To summarize this whole difficult problem one may say that operations on peripheral pathways have but little chance of success."

Many of my patients had undergone intranasal operations, cauterization of the sphenopalatine ganglion, extractions of teeth, injection of medicinal agents into one or more divisions of the fifth cranial nerve, cervical sympathectomies, resection of the lesser and greater superficial petrosal nerves, and ligation of the middle meningeal artery, the temporal artery, and even the external carotid artery—without obtaining complete relief of their pain. A number of interesting observations have been made in these "postoperative cases," but this will have to be reported elsewhere.

Provocative Tests

Failure of treatment and an error in diagnosis are attributable largely to the fact that the physician has not had an opportunity to see the patient in the course of a spontaneous or induced attack of histaminic cephalgia. Therefore provocative tests have been used and figure 4 illustrates the results obtained in a typical case. The reproduction of the attack and its subsequent abortion, although not always possible, are of great value both to the patient and to the physician. They permit the physician and the patient to have a common understanding of the nature of the problem. During the past 15 or more years, I have employed many provocative agents; but, from a practical standpoint, I feel that the subcutaneous administration of 0.35 mg. histamine base is the best agent to employ. This dose will "trip the mechanism" in more than 60 per cent of patients. This is especially true if the patient is experiencing more than one attack each 24 hours. The patient will tolerate this amount without undue discomfort, and larger doses are, as a rule, unnecessary.

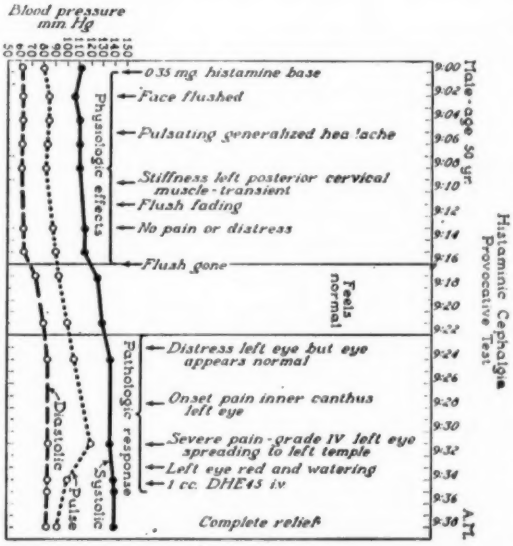

Figure 4. Provocative test which was carried out on October 5, 1950. A 50 year old man had been under observation at the clinic since September 7. During that time, he had had 54 attacks of left histaminic cephalgia and practically all of his attacks occurred at night. The induced attack, graphically illustrated, was identical with his previous spontaneous attacks. After the subcutaneous administration of 0.35 mg. histamine base, the physiologic effects of the drug persisted for 15 minutes. He felt quite normal for six minutes and then the pathologic response of the drug began to manifest itself. He first noted distress in the left eye and the severe pain had reached its height approximately 33 minutes after the beginning of the test. Redness and tearing of the left eye was noted at about the same time, or shortly thereafter. The attack was aborted fairly promply by the intravenous administration of 1 cc. of DHE45. With treatment, the patient's attacks ceased completely within days.

"Histamine headache" and "histaminic cephalgia" should not be regarded as synonymous terms. The immediate reaction after the administration of the histamine, as a provocative test is a generalized histamine headache, which is a physiologic response, lasting about 5 to 10 minutes. This response occurs in normal persons as well as in those who have histaminic cephalgia. Thereafter, for 5 to 30 minutes, and sometimes even longer, after the physiologic effects of the drug have worn off, the patient feels normal. One naturally wonders what is happening during the latent period. Therein may lie the secret to the mechanism of syndrome. In cases of histaminic cephalgia, however, reproduction of the typical attack will occur about 15 to 50 minutes after cessation of the histamine headache and is a pathologic response. It occurs only in cases of histaminic cephalgia. The pain of histamine headache is chiefly intracranial in origin, owing to dilatation of the large vessels at the base of the brain, whereas the pain of histaminic cephalgia results chiefly from dilatation of extracranial vessels.

Histaminic Cephalgia

Histaminic cephalgia appears to represent a localized anaphylactoid reaction with both localized and systemic manifestations. During spontaneous and induced attacks, it seems evident that histamine is released from sensitized cells in the region of the pain and accounts for most of the local phenomena. The rise in the acidity of the gastric contents indicates absorption of this agent in the blood stream, and is comparable to that which is induced by the subcutaneous injections of known amounts of histamine. Other biologic agents may be liberated in the region of pain. The induced attack usually can be aborted promptly by the intravenous administration of 1 cc. of dihydroergotamine (DHE45), which is dihydroergotamine methanesulfonate.

I have frequently been asked, "Does a negative histamine provocative test rule out the presence of histaminic cephalgia?" The answer is no, and an illustrative case follows.

Case #2.—A 46 year old man had his first series of attacks of left histaminic cephalgia in 1942; it persisted for six weeks and subsided spontaneously. He remained entirely free from attacks until the second series began on May 15, 1953. The severe bouts of pain occurred almost exclusively at night, usually ½ to 1½ hours after going to sleep. He occasionally had two or three attacks in one night. "The pain makes me get out of bed," he reported. Pain involved the left upper jaw, left orbital and temporal areas and persisted for 30 minutes to two hours. It required approximately five minutes for the pain to build up to its full intensity and it persisted at that level until it disappeared rather suddenly. During the at-

tack, redness and tearing of the left eye and plugging of the left nostril occurred. There were no trigger areas.

On July 18, he had alcohol injections of the first and second divisions of the left fifth cranial nerve. The attacks ceased for 72 hours and then recurred.

Provocative Tests.—On July 31, the patient was given 0.35 mg. histamine base administered subcutaneously. A negative result was obtained.

Next Day

The next day 0.48 mg. of histamine base was administered subcutaneously and 64 minutes later, a typical attack of left distaminic cephalgia developed. This occurred in spite of the fact that the regions supplied by the first and second divisions of the left fifth cranial nerve were still "numb" as a result of previous injections of alcohol.

Redness and tearing of the left eye were evident and plugging of the left nostril occurred. The patient could not differentiate between the induced attack and the previous spontaneous attacks. The induced attack was aborted within a period of two minutes after the intravenous administration of 1 cc. of DHE45.

I have also often been asked about the value of "histamine skin tests." "Do these tests give the physician accurate information regarding the patient's sensitivity to the drug?" Insofar as my experience is concerned, these skin tests are not of practical value. The size of the flare resulting from the intracutaneous injection of varying amounts of histamine is determined largely by the environmental temperature in which the test is carried out, rather than by the actual sensitivity of the patient. One may, for example, obtain positive results for a skin test in an environmental temperature of 85 to 90 F. and 40 per cent humidity, whereas negative results may be obtained in the same person if the environmental temperature is reduced to 65 to 70 F. and humidity of 40 per cent.

Treatment

Most patients with histaminic cephalgia are incapacitated at the time they come to the clinic. The objective of treatment should be twofold: (1) to alleviate the pain of the acute attack, and (2) to prevent subsequent attacks. In a few cases the breathing of 100 per cent oxygen will alleviate an attack to a marked extent if the attack is mild and if the patient resorts promptly to the use of oxygen. In order for the patient to use oxygen properly, it is necessary for him to have a tank at his bedside with an appropriate mask so that he can use it the moment the attack begins to develop. By so doing, many patients can abort attacks of grades 1 and 2. The use of oxygen, however, is of little or no value in attacks of grades 3 and 4. Severe attacks can frequently be aborted by the prompt intravenous administration of 1 cc. of DHE45 at the onset of the attack or shortly thereafter.

These two procedures may be combined and frequently are. Many physicians seem reluctant to administer DHE45 intravenously, but I have employed it in this manner since our original investigative studies 10 years ago, and I have not noted any ill effects from the use of the drug. If an attack is fully developed and has persisted for several minutes, it is likely to run its course irrespective of the drug employed.

Rectal suppositories which contain appropriate amounts of ergotamine tartrate and caffeine, if used at bedtime, frequently will prevent nocturnal attacks (fig. 3). This is an extremely useful procedure to employ. Suppositories are more effective in preventing nocturnal attacks than is the oral administration of ergotamine tartrate and caffeine.

Treatment of Choice

To prevent future attacks of histaminic cephalgia, histamine desensitization, in my opinion, is still the treatment of choice. My procedure for the subcutaneous administration of histamine is as follows:

I use the contents of a 1-cc. ampule of 0.275 mg. of histamine diphosphate per cubic centimeter, which contains 0.1 mg. of histamine base. Of this ampule, I use 0.05 cc. for the first dose, which is administered subcutaneously. Two injections are administered daily about six to eight hours apart. Each injection is increased by 0.05 cc. until 0.50 cc. is given. In some instances, this amount is not adequate and it is necessary to continue to increase the amount until 0.75 cc. or even 1 cc. is reached. Adequate dosage is determined by the patient's response. If immediate flushing of the face or throbbing is not experienced with the injection and if the patient's symptoms decrease in severity or frequency, the procedure is adequate. However, if the symptoms are aggravated or seem to be precipitated by the injections, the patient may be undergoing sensitization rather than desensitization. In such a case, the subsequent dose is reduced by half and, again, the amount administered at each injection is increased by increments of 0.05 cc. to a level just below the amount which precipitated the symptoms. This total amount is then injected twice daily as the maintenance dose. The patient is instructed in the technique of administering the injections to himself, whether the dose is 0.25 cc. or 0.75 cc., and is told to continue to use them twice daily at home for two or three weeks and once daily thereafter for an indefinite time. Some patients find it necessary to continue to use one injection, or even two or three injections, daily at home in order to remain free of symptoms. The maintenance doses of histamine may be continued for a life-time, if necessary, without harm. I am convinced that the use of smaller amounts of histamine at more frequent intervals controls symptoms more satisfactorily than large amounts administered subcutaneously only once daily or every other day.

False Security

After a long period of freedom from symptoms, many patients are lulled into a false sense of security, and they discontinue the injections of maintenance

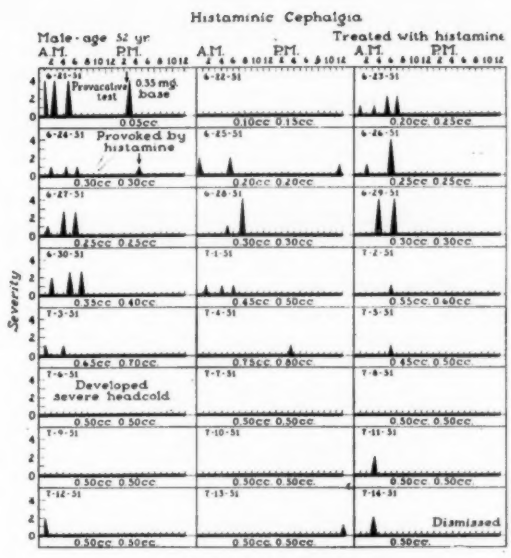

Figure 5. Graphic record illustrating clinical course and dosage of histamine employed in the treatment of the third series of attacks of histaminic cephalgia in a man aged 52 years.

doses. Many others report a recurrence of symptoms coincidental with an infection of the upper part of the respiratory tract. The maintenance dose then is inadequate, and resuming the desensitization schedule is often difficult because of the change in the patient's sensitivity. Many patients with histaminic cephalgia who experience a recurrence of headaches, report that they have attempted to resume the desensitization schedule and to increase the dose to the former maintenance level without realizing that, with a recurrance of the attacks, their sensitivity to histamine is greater than it was during the previous episode. The reason for this has not been determined. Only careful reports and observations of the patient's symptoms and response to injections in each 24 hours will give a clue to the schedule most satisfactory for each patient; hence, my expression that each schedule has to be "tailor-made" to fit the individual patient.

Histamine Desensitization

It is usually easy to treat a patient with histaminic cephalgia when he is observed for the first time, provided he has not previously had histamine therapy. About 95 per cent of the patients will respond to histamine desensitization, and the attacks, irrespective of their severity, frequency and duration, will cease within 10 to 20 days. However, if the patient has had histamine therapy previously, and especially if the dose has been above his reaction level, attempts to repeat the procedure cause the problem to become increasingly difficult.

The following brief report of a case illustrates this phase of the problem.

Case #3.—A man, aged 52 years, having three to four nightly attacks of left histaminic cephalgia for two years, came to the clinic in June, 1951. He was totally incapacitated because of the frequency and severity of his attacks. All attacks involved the left side of the head and tended to occur about one and one-half hours after he had gone to sleep. The pain, which was sudden in onset, persisted for approximately 30 minutes and terminated promptly. The pattern repeated itself whether he slept at night or during the day. At the time of his registration at the clinic he was suffering the third series of attacks, all of which had been experienced within the previous six years. The patient came as an emergency case and was accompanied by his wife and a trained nurse.

Previous Treatment.—In March, 1948, histamine desensitization was initiated and resulted in relief from attacks for eight months. Again, in November, 1948, histamine desensitization was carried out and this time relief was obtained for five months. Each time a maximal dose of 1 cc. standard solution, that is, 0.275 mg. histamine diphosphate per cubic centimeter, was abministered. In April, 1949, histamine desensitization was carried out for a third time. The same dosage was used. However, the attacks became more frequent and more severe, indicating that the patient was being "sensitized" to the drug.

In May 1949, the intravenous administration of histamine precipitated a severe attack. "I thought I would go wild with the pain," stated the patient.

Section of the left occipital nerve was performed within the month but the patient did not obtain relief.

Provocative Test.—The results of our standard histamine provocative test were positive. A typical attack of left histaminic cephalgia developed within 34 minutes after the subcutaneous administration of 0.35 mg. histamine base.

The treatment and the clinical course are depicted in figure 5. The patient was instructed to continue the use of 0.50 cc. of histamine daily, and has remained well.

To treat a succeeding series of attacks of histaminic cephalgia, the dose employed is decreased consistently. Experience has shown that if the same dose is used while treating the third series of attacks as was previously employed in the first, the patient may be made worse instead of better.

Histamine therapy is neither simple nor commonplace. It is fraught with many pitfalls. It is difficult to outline a schedule which will fit any large group of patients. The schedule *must* be "tailor-made" to fit each patient. Haphazard histamine therapy may produce haphazard results. Histamine is the most potent agent, so far as I am aware, known to medical science. It is up to the physician to employ it wisely.

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(Continued on page 167)

Treatment of Migraine* and Tension Headache

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It has been estimated that more than half of all patients who seek medical advice or attention have headaches as one of their complaints. In this vast majority the symptoms are either caused by migraine or are primarily associated with an emotional disturbance (tension or muscle contraction headache).

Rational therapy starts with the prerequisite that the physician should be able to diagnose the presenting type or types of headache. To make a diagnosis the physician should understand what is known about the underlying physiologic and psychologic mechanisms associated with headache. In addition, of course, a detailed history and thorough examination are essential.

In the management of migraine and tension headache (muscle contraction headache), one is dealing with both physical and emotional factors. Therefore, the goals of therapy are limited by the potentialities of the patient, the pharmacologic actions of the medications used, and the physician's interest in and orientation toward the problem. It has been my experience that a dual approach using pharmacotherapy and psychotherapy is the most successful method of therapy.

Pharmacotherapy: In pharmacologic treatment one or more of the following is attempted: (1) to raise the pain threshold, (2) to interrupt the mechanism of producing pain, or (3) to reduce the emotional tension and anxiety associated with the pain (fig. 1). However, the efficiency of any drug does not depend upon its pharmacological action alone. The importance of dosage, timing, mode of administration and toleration by the patient cannot be stressed sufficiently. Furthermore, such efficiency greatly depends upon the patient-physician relationship, which includes among other things, the length and frequency of interviews with the patient. Our experience indicates that the patients' reactions to drugs not only may depend upon the underlying disorder responsible for the headache, but also upon the degree of incapacity, constitutional makeup of the patient, duration of symptoms, age of individual and psychological status. What the taking of medication symbolizes to the patient is also a contributory factor in treatment. Is the taking of medication a sign of weakness to the patient to which he might resist, of receiving omnipotent mystical power, or of love and affection? All these factors must be given serious consideration in clinical usage of drugs in treatment of headache.

DRUGS USED IN TREATMENT OF HEADACHE

ANALGESICS

SEDATIVES

STIMULANTS OF THE CENTRAL NERVOUS SYSTEM

DRUGS ACTING ON THE CIRCULATION

HISTAMINE AND ANTIHISTAMINE AGENTS

VITAMINS

HORMONES

AUTONOMIC NERVOUS SYSTEM
(ACTING ON AUTONOMIC EFFECTOR CELLS)
CHOLINERGIC
ADRENERGIC

FIGURE 1.

In evaluating the subjective symptoms of head pain, there is such a tremendous psychologic factor that great care must be used in reporting success with any type of treatment. Investigation of the literature and of some of our own work revealed that good results obtained from the primary investigators are too infrequently duplicated on subsequent tests.

Psychotherapy: Psychotherapy is directed toward relieving the emotional tension and stress that are the precipitating factors in an attack of tension headache. The most important single factor in the psychotherapeutic process is the emotional relationship of the patient to the physician. This relationship dominates the treatment, despite the fact that it may be explicity discussed only rarely or not at all. In complete psychoanalysis, of course, this relationship is clearly recognized by the therapist.

In emphasizing the importance of the physician-patient relationship, one must not overlook the importance of other aspects of psychotherapeutic procedure such as environmental manipulation and the ventilation of wholly or partly unconscious emotional conflicts by the patient.

In environmental management one has to recognize and deal with the patient's capacity to handle his work, recreation, family and social life. The physician should strive to correct the patient's program so that work, play and rest are balanced. In some cases a sustained hobby is of value in securing relief from tension and daily stress. Concerning any changes

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in the patient's way of life, it is important that the doctor assist the patient in making his own decisions rather than making them for him.

Often simple explanations of the effect emotions have on bodily functions and the relationship of stress situations to development of symptoms may give the patient insight into what occurs to him during periods of stress. Reassurance, suggestion, and reeducation are other helpful technics.

Such therapy is within the province and ability of the interested general practitioner. With this treatment, over-all improvement in terms of greater capacity for life adjustment and a reduction of the habit patterns that lead to activation of the physiologic mechanism causing the headache can be anticipated. Formalized psychiatric treatment is necessary for some patients in whom reorganization of the personality is necessary to secure relief.

Migraine Therapy

Symptomatic: Pharmacotherapy has been markedly successful in the management of an actual attack of migraine. At present the ergot derivatives are the most useful drugs in the treatment of a migraine attack and the most effective of these alkaloids is ergotamine tartrate. Clinical evidence of its beneficial effects has been supported by experimental observations in the laboratory.

Ergot is a fungus (*Claviceps purpurea*) growing in the grain of cereals and is obtained from rye. All ergot alkaloids are derived from lysergic acid and are related chemically as well in their actions.

The active levorotatory isomers of all ergot alkaloids posess powerful central nervous system actions, while those ergot alkaloids containing polypeptide side chain (i.e., ergotamine and ergotoxine) also have a significant adrenergic blocking activity.

One very characteristic effect of the ergot alkaloid is a direct stimulation of smooth muscle in many organs. In most cases it occurse in smaller doses than necessary to produce adrenergic blocking.

Although ergot alkaloids with significant adrenergic blocking activity contain a polypeptide side chain that is noted in ergotamine and ergotoxine, it is questionable whether this effect takes place in the dosage used in the treatment of headache. Hydrogenation decreases the ability to stimulate smooth muscle and increases the adrenergic blocking activity of all natural alkaloids. In the treatment of migraine the hydrogenation of ergot alkaloids that increases adrenergic blocking activity reduces the effectiveness in migraine treatment.

The benefit that ensues following the taking of ergotamine for an attack of migraine is probably the result of the direct action of the substance on the smooth muscles of the blood vessels, producing a constriction of these vessels that causes a decrease in amplitude of pulsation. This is particularly noticeable in the circulation of the external carotid.

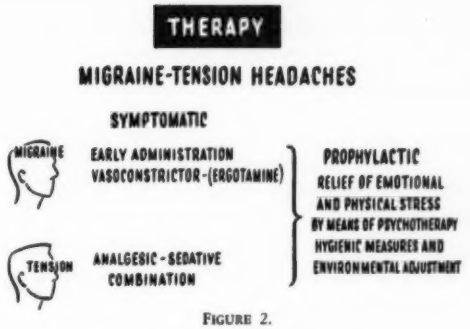

FIGURE 2.

Symptomatic Treatment

Symptomatic treatment of migraine is most effective by use of oral or rectal ergotamine tartrate and caffeine. Nausea and vomiting can be relieved by use of an antispasmodic and/or sedative with this preparation. Rectal use has proved empirically to be most efficacious, especially when oral medication cannot be retained (fig. 2). It is likely that rectal medication has the advantage of being absorbed more directly into the systemic circulation without having to penetrate the hepatic and gastric barriers. Hence, it is postulated that its action is faster with fewer side effects than by oral administration. This requires further investigation.

The importance of administering the medication early in the course of an attack, as well as in adequate doses, cannot be over-estimated. Many failures in therapy are caused by an inadequate dose given too late. The optimal time of administration is in the prodromal period or immediately after onset of the headache.

Ergotamine is contraindicated in organic heart disease, obliterative vascular disease, hypertension, pregnancy, hepatic disease and septic states associated with intravascular foci.

In some patients ergotamine tartrate is poorly tolerated and the use of dihydroergotamine methanesulfonate (DHE-45) is indicated. Dihydrogenation of ergot reduces the vasoconstrictive action. In order to produce an equivalent therapeutic effect, dihydroergotamine must be given in doses twice as great as ergotamine tartrate. It can only be administered parenterally, and like ergotamine, must be given as early as possible in the attack.

Vasodilators such as histamine, B-methylacetylcholine (mecholyl), nicotinic acid, amyl nitrite, intravenous magnesium sulfate, and so on, have been used in the prodromal stage (vasoconstrictor phase) in order to abort the attack, and in the headache stage (vasodilator phase) to lower the blood pressure sufficiently to reduce the arterial pulsations. Except in a few isolated cases, these preparations are of little value.

Analgesics and sedatives are sometimes indicated if the headache has been present long enough for edema to take place and the vessels to become firm

and tortuous. In these cases codeine phosphate with or without sodium amytal or seconal (0.1 gm.) is indicated.

Prophylactic: It is well known that suitable psychotherapy may be of great value in reducing the intensity and number of migraine attacks in most patients, and may even result in their disappearance for a considerable time. The details of this type of therapy have already been discussed.

Adequate relaxation, improvement in sleep and correction of any physiological abnormalities will be of help in reducing the frequency of the attacks. In a few cases there may be evidence that the precipitating mechanism is allergic, endocrine or "metabolic." It has been my experience that these factors are too isolated to be of real value in therapy. However, when an offending allergen is responsible it should be removed or the patient desensitized. If the onset of migraine is associated with the menstrual period the use of progesterone or testosterone, or both, may occasionally be helpful.

Reported studies in patients with migraine and other types of vascular headache indicated that there are major alterations in electrolyte balance in association with headache. These are characterized by low rates of excretion of water, sodium, potassium and creatinine prior to headache and higher rates of excretion during the phase of subsidence of headache. In the premigraine phase and early stages of an attack, the sodium and other electrolytes rise to a high level, while at the same time there is a marked hydration of blood. The suggestion is made that these disturbances are caused by disordered functions of the hormones governing salt and water metabolism of the adrenal cortex and posterior pituitary gland and that in women these are exacerbated by the influence of certain sex hormones.

Hydrated Patients

In our investigative work we have hydrated patients with intravenous infusions after which an antidiuretic hormone and later a diuretic hormone were administered. No abnormal findings were obtained in any of these patients and none developed headaches subsequent to the tests. It is most likely that fluid electrolyte and renal changes are widespread evidence of bodily changes accompanying vascular headaches and not a causal factor.

In those patients in whom rapid weight gain and afterwards polyuria occur, the use of various diuretics is worth a trial. Among these are the acid diuretic salts, such as ammonium chloride, urea, and a newer group of diuretics, which have been recently introduced, such as neohydrine and diamox. Our experience with diamox in a small number of patients has been reasonably successful. However, these indications are not final proof of the value of this type of medication. We still do not know whether a diuretic that works primarily on the renal tubules or one that prevents or suppresses the A.D.H. action of pitressin is the most valuable in treating headaches associated with electrolyte imbalance.

Drugs Of No Value

The use of such drugs as histamine, nicotinic acid, and so on, in my experience is of no value. Sedatives to reduce emotional tension are limited in their usefulness. Recently we have used reserpine and thorazine as interim treatment with migraine patients; our results have not been encouraging.

The periodic and often paroxysmal nature of migraine and other types of vascular headache brought forth the idea that treatment with anticonvulsants might be of value in these disorders.

A small number of migraine sufferers respond well to mesantoin and dilantin prophylactically. This limited group consists of patients who have an aura of aphasia, paresthesias or hemiplegia prior to the headache and an abnormal encephalogram. In certain other patients who have a family history of migraine and epilepsy and on encephalogram show an abnormal brain wave with spiked patterns, the use of certain anticonvulsive drugs is of value in reducing the frequency, severity and duration of the headache.

The hydantoins are closely related to the barbiturates, being derivatives of glycolyl urea as compared with malonyl urea. Both hydantoins, dilantin and mesantoin, may produce bizarre and unsuspected side reactions which occasionally may limit their usefulness in therapy. Of special interest is the effect of mesantoin on the hematopoietic system, which may produce an increase in lymphocytes and depression of the polymorphonuclear leukocytes. Hence it is necessary to follow these drugs with blood counts that can be the earliest indication of toxicity. There have been cases of aplastic anemia and leukopenia following the use of mesantoin reported in the literature.

Tension Headache

Symptomatic: Symptomatic relief from tension headache is secured by drug therapy, which as in psychotherapy, must have flexibility for its keynote. The treatment of tension headache is nonspecific, but the purpose of therapy is to relieve tension and raise the pain threshold. This is best accomplished by use of an analgesic-sedative combination. Such a combination should react with minimal side effects allowing the patient to maintain his environmental relations and not become a problem because of addiction potential or development of tolerance.

The most practical and useful analgesics used in the treatment of headache are the antipyretic analgesic group of coal tar derivatives, such as acetylsalicylic acid and acetophenetidin. They act by raising the pain threshold possibly through depression of pain centers in the thalamus. In therapeutic doses they do cause mental disturbances, anesthesia or changes in modalities of sensation. The types of pain relieved by salicylates are of low intensities.

Certain techniques, particularly those of Hardy and Wolf, demonstrated that acetylsalicylic acid elevates the pain threshold half as much as morphine. Other investigators have not been able to demonstrate threshold raising action in animals or man. All analgesics have a ceiling beyond which no additional increase in the pain threshold is obtained. A maximum analgesic action may be secured by relatively small doses such as 10 grains of acetylsalicylic acid or acetophenetidin. However, the duration of action is prolonged by more frequent doses. Combinations of analgesic drugs do not increase the maximum effect, the ultimate threshold being that of the most active component (i.e., codeine and acetylsalicylic acid pain threshold is raised only to that of codeine). Analgesics do not potentiate the hypnotic action of barbiturates but barbiturates potentiate the action of analgesics.

The most effective symptomatic medication in the treatment of tension headache has been several analgesic and sedative combinations. In some cases empirin compound will suffice.

Opiate analgesics are used for the relief of severe pain as well as a sedative action. However, we do not recommend the use of morphine or synthetic drugs like demerol, methadon, and so on, in the treatment of chronic headache because of their addiction potentialities.

Codeine (methylmorphine) occurs in opium to the extent of about 0.5 per cent. It possesses much less analgesic potency than morphine but is the most effective relatively safe analgesic drug for treatment of chronic headache. Although both tolerance and addiction from continuous use have been recognized, these are uncommon.

Prophylactic: Control of tension headaches can best be accomplished by use of psychotherapy, for

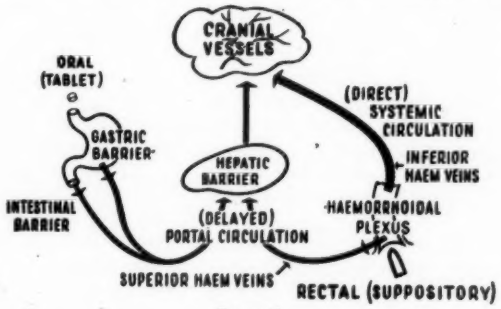

FIGURE 3.

this is the only method in which the patient's emotional conflicts can be resolved.

The use of sedatives such as barbitals and analgesics are only of temporary effect. Furthermore, their side effects curtail prolonged usage.

More recently, we have used reserpine on over 100 patients with tension headaches. They have been treated for periods of 2 ot 9 months with moderate success. Improvement was noted in 2 out of 3 patients on the average dosage range of 0.75 mg. daily. Little difficulty was encountered from side effects. However, as has been noted elsewhere, the effectiveness of any drug with headache patients consists of a multiplicity of factors. The results with reserpine should be considered only as a preliminary trend and not conclusive.

Conclusions

Migraine and tension headache should be treated both symptomatically and prophylactically (fig. 3). Symptomatic treatment is essentially one of pharmacotherapy while prevention of an attack is best accomplished by psychotherapy.

Clinical Notes From Grand Rounds

These notes are abstracts of opinions expressed by staff members during case presentations at the Friday Grand Rounds of the Pratt Diagnostic Clinic — New England Center Hospital.

Iceland disease is the name of a new epidemic disorder that is sometimes confused with poliomyelitis and polyneuritis. No etiologic agent has as yet been isolated. It was first described in Iceland. Epidemics have also occurred in Florida and New York State, and isolated cases have been encountered at many other points. Clinically it is characterized by fever, headaches, muscle aches, tenderness, ensory disturbances (numbness and dysesthesia), and emotional instability. Pain may occur in the distribution of a nerve root. Abortion commonly occurs in pregnant women. The spinal fluid findings are variable; they consist largely of an increase in lymphocytes. Occasionally, splenomegaly may be present. The course is often prolonged, symptoms continuing at times up to 15 months, consisting largely of fatigue and body pains. The prognosis for eventual recovery is good.

▼ ▼ ▼

In from 10 to 40 per cent of patients shortly following mitral commisurotomy fever, chest pain, arthralgias, transient pleurisy, and electrocardiographic changes (inverted T waves and prolonged P-R interval) occur. It is believed that the stress of the operation reactivates rheumatic fever. The symptoms respond to salicylates or subside spontaneously, but may recur and again respond to treatment.

(Continued from page 163)

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Neurosurgical Aspects of Head Pain

By Bertram Selverstone, M.D., Neuro-surgeon-in-Chief, New England Center Hospital; Professor of Neurosurgery, Tufts University School of Medicine, Boston

The neurosurgeon has a twofold interest in the treatment of head pain. He is on surest ground when his purpose is the removal of a lesion that is causing pain. He must tread with more caution when he attempts to relieve pain by interrupting its pathways to consciousness, since he must usually accomplish his objective by producing some degree of neurological deficit. A small area of sensory loss that would be accepted gratefully as a substitute for pain will add to a patient's distress if even a part of the original discomfort persists after operation.

Most of the soft tissues of the face and scalp, as well as the deeper structures of the pharynx and neck, contain nerve endings for pain. The structures within the skull, as Wolff and Ray have so carefully shown, are sensitive to pain only in certain specialized regions.

Outside Origins

It is not surprising that a great number of pains in the head and face have their origins outside the skull in branches of the external carotid artery, for example, and in inflammations and tumors of the scalp, paranasal sinuses and orbits. Carcinomas and other malignant lesions in these areas may present distressing problems to the neurosurgeon. A well organized tumor clinic, where the neurosurgeon works with the otolaryngologist, oral surgeon, ophthalmologist, general surgeon and radiologist, provides an ideal setting for evaluation and treatment of these patients.

When such tumors invade the central nervous system or its coverings, the neurosurgeon must attempt their removal. Invasion of cranial nerves or of other painful structures makes it necessary for him to interrupt pathways in the fifth, seventh, ninth and tenth cranial nerves, and in the upper three cervical nerves, for the relief of unbearable pain in the head and face.

Complex Procedures

But section of sensory roots may, for reasons that are far from clear, fail to relieve the pain of a deeply invasive malignant lesion, and the neurosurgeon may be forced to attempt more complex procedures upon the central pain pathways. Elsewhere in the body, we know that interruption of the second pain neuron by anterolateral cordotomy is most effective for relief of pain in the limbs or trunk, even though caused by a highly invasive cancer.

For relief of head and face pain, we may interrupt this second neuron in the quintothalamic path of the mesencephalon or may destroy its end station in the posteromedial ventral nucleus of the thalamus. Such procedures as these, whether performed by means of standard neurosurgical methods or by stereotaxic techniques, are already with us but they are as yet neither safe enough nor sufficiently reliable for routine use. More often than we should like, we are forced to use one of the varieties of prefrontal lobotomy which, without interrupting its pathways, may alter emotional responses to pain and also to impending death.

Dramatic Relief

Pain originating in the blood vessels of the scalp and face may come to the attention of the neurosurgeon either before or after medical means for its management have been attempted. The dramatic relief that arterial section brings to the patient with cranial arteritis must not lead to fruitless efforts to treat vascular headaches such as migraine routinely by division of branches of the external carotid artery.

There is no doubt that vascular ligation may provide relief from painful vasodilatation for weeks or months, but pain may recur worse than before when new collaterals dilate. Occasionally, a patient with unusually severe migraine becomes addicted to morphine or demerol or threatens suicide. In such a case, the neurosurgeon may justifiably divide the medial fibers of the trigeminal posterior root, and will usually relieve the pain.

Increased Pressure

Within the skull, we have often thought of increased intracranial pressure as a cause of head pain. It has become increasingly clear that symmetrically increased pressure may produce no pain at all. The brain and most of the pia and arachnoid, except where great vessels enter or leave, are insensitive as is most of the dura over the vault of the skull. The basal dura, falx and tentorium and the meningeal and large basal arteries are sensitive, as are the walls of the great venous sinuses.

A brain tumor or a subdural hematoma may produce severe headache without increasing the intracranial pressure. It does so by distorting or pressing upon the pain-sensitive intracranial structures. In a meningioma, dilatation of the meningeal vessels which help to feed it may be painful. Sensory cranial nerves may also be compressed or stretched by basal meningiomas. After removal of a brain tumor, headache is usually immediately relieved, although increased intracranial pressure may persist for several days. Return of a deformed sensitive structure, such as a stretched internal carotid or middle cerebral artery, to normal position is presumably responsible for relief of the pain (fig. 1).

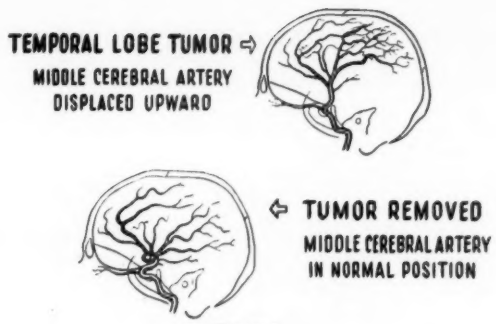

FIGURE 1.

Cerebral Angiography

With the advent of cerebral angiography, which usually permits precise location of an aneurysm or vascular malformation, the neurosurgeon has seen increasing numbers of patients who have had a recent subarachnoid hemorrhage. When an aneurysm ruptures into the subarachnoid space, it produces an excruciating, sudden pain as the arterial stream distorts and deforms the great vessels about the base of the brain. If the patient survives the initial catastrophe, as he usually does, headache persists as long as blood and its breakdown products irritate basal meninges and sensory nerve roots, both cranial and cervical.

Aneurysms may also produce pain before they actually rupture. Vague, poorly localized headaches may result from distention of the walls of an enlarging aneurysm, but much more severe and acute pain comes when an aneurysm impinges against one or more branches of the trigeminal nerve as they pass into or through the cavernous sinus. Jefferson has shown that from the location of the characteristic pain and numbness, the precise site of an intracavernous carotid aneurysm may be localized.

Pain caused by aneurysmal compression of trigeminal branches may be momentarily relieved quite completely by compression of the common or internal carotid artery in the neck. If the aneurysm is clipped or trapped between clips or, indeed, if the internal carotid artery is simply occluded in the neck, pressure within the aneurysm is so reduced that the pain is permanently relieved (fig. 2).

Certain head and face pains are dramatically relieved by interruption of pain pathways. In this group is *tic douloureux,* the common paroxysmal neuralgia of the trigeminal nerve. While we are not certain of the site of the pathological lesion responsible for the sudden, unilateral, lancinating pain of this disease, it seems likely that it lies in the Gasserian ganglion or in its posterior root. Since pain is temporarily relieved, sometimes for years, by injection or section of peripheral branches of the nerve, it is likely that denervation of the "trigger zone" rather than of the painful area itself is responsible for the good effect of these procedures.

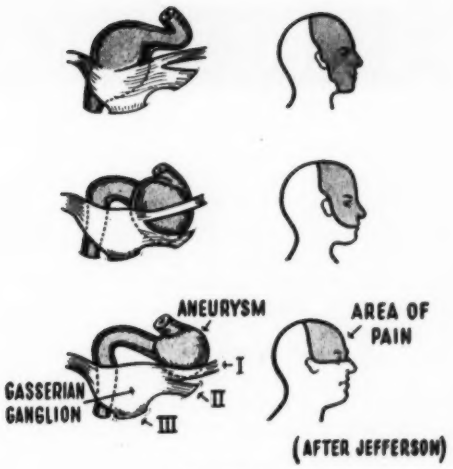

FIGURE 2.

There are less common neuralgias of the nervus intermedius and of the glossopharyngeal nerve which seem quite analogous in their clinical features to *tic douloureux.* These are all permanently relieved by section of the appropriate posterior roots. Our success with such operations should not encourage their indiscriminate use in the "atypical" neuralgias of the face and head, especially those characterized by constant aching, throbbing or burning pain. Denervation of a painful area is by no means a cure-all. In postherpetic neuralgia, for example, posterior root section is fruitless, since the disease involves the *substantia gelatinosa* of the trigeminal nucleus itself as well as the sensory cells of the Gasserian ganglion.

Syndrome Described

Since section of the second neuron by means of anterolateral cordotomy will effectively relieve postherpetic neuralgia of the trunk or limbs, we believe that we shall eventually be able to treat it in the face and head by producing lesions in secondary trigeminal paths in midbrain or thalamus. The syndrome so well described by Horton as "histamine cephalalgia," where paroxysms of head and face pain are associated with unilateral lacrimation and conjunctival injection, can sometimes be treated surgically. A number of such patients, as reported by Gardner, may be helped by section of the greater superficial petrosal nerve. We have relieved a number of patients with severe attacks of this type by radical devascularization of the meninges in the middle cranial fossa. Most of them are, however, best treated by nonsurgical methods.

It is the neurosurgeon's first responsibility to sort out from the great number of patients who complain of head pain those who have potentially dangerous lesions. Among the rest, he must choose carefully, for an ill-conceived operation, even a minor one, may be the first step of a fruitless Odyssey in which the patient travels from clinic to clinic, seeking an external solution for a problem whose answer, if there is one, lies within himself.

CURRENT THERAPY

Agammaglobulinemia

By Jack A. Bernard, M. D., El Paso

In recent years, there has been described in adults and children a syndrome in which recurrent severe infections, commonly pulmonary, have occurred in association with marked depression or absence of gamma globulin in the serum. (In infants: Intractable diarrhea and sudden death). This seemingly rare condition warrants publicizing because its recognition is important in that such cases may be treated with gamma globulin thereby reducing morbidity and prolonging life.

Agammaglobulinemia is characterized by marked depression or absence of gamma globulin, multiple infections and inability to form antibodies. The infections are mostly bacterial and include pyoderma, oteitis media, pneumonia, sepsis, and others. Normal viral antibody mechanisms are thought to be intact but one case of fatal generalized vaccinia is reported.

Pathological Sections

Pathological sections have shown only diminished gamma globulin content of the lymphoid tissue and the absence of plasma and preplasma cells.

Most of the cases described have occurred in children and have been predominately in males. These had recurring infections since infancy. The infections have responded promptly to antibiotics but would return promptly after therapy was discontinued.

There seems to be two types of agammaglobulinemia: One in which the patient is rarely ill, has edema with hypoproteinemia, and does not respond to various antigens, but responds to high protein feedings. The other type: In which the absence of gamma globulin is accompanied by recurrent infections and there is lack of response to toxoid or antigens and where there is response to injections of gamma globulin. The latter type has been reported more often in young boys (age 8 or 9) and is thought perhaps to be congenital in origin. Also there is a similar acquired type in adults, commonly associated with malignant lymphomas and other blood dyscrasias.

Susceptibility to Infection

In the cases of pure gamma globulin deficiency, the predominate clinical feature is susceptibility to infection. Such cases probably did not survive before the days of antibiotics. Hence this and the previous lack of methods of estimating gamma globulin may explain why such cases have not previously been described.

Diagnosis may be suggested by the history of repeated infections with the same bacterial or viral agent, absent or low immune response to vaccination or innoculations, such as the Schick and Dick tests and Tuberculin test.

Early Diagnosis

Much remains to be clarified as regards agammaglobulinemia. Early diagnosis is essential as such patients may be benefited, perhaps kept free from infection, with prolongation of life, by monthly subcutaneous injections of 0.1 gm. gram of pooled normal serum gamma globulin per kilogram of body weight. If no response, prophylactic antibiotic therapy alone or in conjunction with the gamma globulin may be helpful.

The physician should be alert for this apparently rare deficiency in children, particularly males who have had repeated respiratory or other infections, and in others who have had repeated infections, "poor resistance," or poor response to immune reactions. To confirm the diagnosis the blood may be taken from such suspected cases and the serum forwarded to the laboratory for study.

Course In Surgery In Acute Trauma At William Beaumont Hospital

A course in surgery in acute trauma will be conducted at William Beaumont Army Hospital, El Paso, April 2-6.

This course covers the important aspects and methods of handling patients subjected to various types of trauma. It includes consideration of such subjects as triage, the shock state, control of hemorrhage, debridement, management and transportation of the wounded, principles of treatment of bone and joint injuries, abdominal and chest injuries, head injuries, etc.

The course is open to civilian as well as military physicians without payment of a fee. Any civilian physician desiring to attend is advised to write to:

Commanding Officer
William Beaumont Army Hospital
El Paso, Texas

Reserve officers, not on active duty, desiring to receive credit points for attendance, are advised to contact their local Army Reserve Office or Military District Headquarters.

Military physicians are advised to apply through their Commanding Officer.

APHORISMS and MEMORABILIA

Miscellaneous Truths and Concepts

By Andrew M. Babey, M.D., Las Cruces, N. M.

1. "To greet a patient with, "You look better," without even having really looked at him, is harmful." Edward A. Strecker; *Veterans Administration Tech. Bull.* April 15, 1954; Section 10-99 P. 3.

2. "Of all the techniques of psychotherapy, suggestion is the least useful . . . . Suggestion smacks too much of the ancient medicine men. It is illogical. It is just as erroneous for the patient to believe that stroking the palsied arm or a "shot" of electricity cured the paralysis as was the original belief that the arm was physically paralyzed. The appeal of suggestion is to emotional immaturity and it encourages emotional dependencies. Finally, it is inherently dishonest, and it threatens the relationship between patient and doctor which must be built upon mutual frankness and confidence." E. Strecker; *loc. cit.;* P. 4

3. "Indiscriminate counseling is harmful. It creates unmanageable emotional dependencies and weakens the ego. Decisions should not be made for the patient about such weighty matters as marriage, divorce, occupation, etc." E. Strecker; *loc. cit.;* P. 5

4. "The physician who hates Jews, Negroes, or Catholics, or Methodists, New Dealers, Republicans, Democrats, or, indeed, whose hatred of those who are not like him or do not think as he does, is not fit to deal with human emotional problems." E. Strecker; *loc. cit.;* P. 6

5. "The wise therapist will be too wary to take on both husband and wife for continued treatment. He will be subjected to too strong an insistence that he take sides." E. Strecker; *loc. cit.;* P. 7

Necessity of Religion

6. "In 1932, Jung wrote, "During the past thirty years people from all civilized countries of the earth have consulted me. I have treated many hundreds of patients, the large number being Protestants, a small number of Jews, and not more than five or six believing Catholics. Among all my patients in the second half of life . . . there has not been one whose problem in the last resort was not that of finding a religious outlook on life. It is safe to say that every one of them fell ill because he had lost that which the living religions of every age had given their followers, and none of them had been really healed who did not regain his religious outlook." E. Strecker; *loc. cit.;* P. 8

7. "Therapy is a two-way process. Not only is the therapist making an image of the patient, but the patient is constructing a facsimile of the therapist. And the patient is psychologically uncanny in noting signs of disinterest, boredom, sentimental sympathy, antagonism." E. Strecker; *loc. cit.;* P. 9.

8. "It is abundantly clear that the blood stream is endowed with powerful antibacterial defenses and that, in most instances, the ability of the circulation to clear itself of invading pathogenic organisms persists unimpaired even in the dying host." Ivan L. Bennett; *Veterans Administration Tech. Bull.* November 30, 1954; Section 10-104 P. 7

9. "The poor prognostic significance of bacteremia in the course of infections is not at all due to multiplication of bacteria in the circulation but stems from its usefulness as an index of failure of localization of the infectious process in extravascular tissues, allowing spread and entry into the blood stream." I. Bennett; *loc. cit.;* P. 7

Time Relationship

10. "The time relationship between bacteremia and chills is important to bear in mind. Following release of microorganisms into the blood, there is ordinarily a *lag-period* of 1 to 2 hours before the chill and rise in temperature begin. Because bacteria are usually cleared from the circulation very rapidly, the blood can become sterile long before the onset of the rigor and blood cultures obtained during a chill are often misleadingly negative." I. Bennett; *loc. cit.;* P. 7

11. "Despite a widespread tendency to regard bacterial invasion of the blood in endocarditis as consisting of irregular spurts and showers of organisms occurring when valvular vegetations break off, bacteria appear to enter the circulation at a constant rate." I. Bennett; *loc. cit.;* P. 10

12. "It is striking that there is practically no middle ground in obtaining positive blood cultures; the situation in most cases is either that the causative organism is easily obtained or else consistently negative results greet even the most intensive efforts. Cultures are often all positive or all negative; intermittently positive cultures are uncommon. After reviewing the available evidence on delaying therapy in bacterial endocarditis, Finland concluded that a short waiting period does not impair the end result in uncomplicated cases. It can be stated with a fair degree of certainty that the length of this delay should not exceed 5 days and that an even shorter period may be optional." I. Bennett; *loc. cit.;* P. 10

13. "Because of the possibility of infection with anaerobic or microaerophilic strains, thioglycollate broth should always be inoculated at the same time aerobic cultures are made. This is particularly important on gynecological services. The practice of making agar pourplates at the bedside is certainly acceptable, but the writer has not been impressed

with its usefulness and it unquestionably makes the taking of cultures more tedious and time-consuming." I. Bennett; *loc. cit.;* P. 10

Arterial Blood Suggested

14. "When ordinary venous blood cultures show no growth, the suggestion to obtain arterial blood is sometimes made. Despite occasional statements advocating this procedure, the balance of clinical and experimental evidence indicates that there is no removal of bacteria as blood circulates through the upper extremities. Consequently, blood from the antecubital vein is as likely to contain organisms as is blood from the femoral or brachial arteries." I. Bennet; *loc. cit.;* P. 11

15. "The characteristic lag-periods between entrance of bacteria into the circulation and the occurrence of chills has been mentioned. Consequently, in patients with intermittent chills or with spiking fever without rigors, blood for culture should be drawn during the hour or 2 preceding an expected temperature rise." I. Bennett; *loc. cit.;* P. 11

16. "The preceding discussion of bacteremia in endocarditis makes it evident that a schedule for diagnostic blood sampling in this disease consisting of 2 to 4 cultures at covenient intervals during each day is fully adequate and far more reasonable than the practice of taking cultures at intervals of 1 to 2 hours around the clock which is often advocated in a mistaken effort to "catch the organism."" I. Bennett; *loc. cit.;* P. 11

17. "It is fairly common to make colony counts from agar plates of blood cultures . . . . few conclusions can be drawn from colony counts." I. Bennett; *loc. cit; P.* 11

Bone Marrow Culture

18. Occasionally, *bone marrow culture* gives results superior to those obtained from peripheral blood. This is likely in typhoid after the first week, in some cases of brucellosis and subacute endocarditis, and in histoplasmosis. The advantage of marrow culture in these diseases may be due to the survival of the causative organisms in macrophages of which marrow is a rich source." I. Bennett; *loc. cit.;* P. 11

19. "Meningococci can sometimes be demonstrated in smears of tissue juice (not capillary blood) obtained from skin lesions. The lesion should be squeezed between thumb and forefinger and then punctured with a sharp blade to obtain fluid for smear and culture." I. Bennett; *loc. cit.;* P. 11

20. "In rare instances, direct observation of a stained smear of peripheral blood or buffy coat reveals organisms. This is commoner in meningococcemia than any other disease and should always be attempted." I. Bennett; *loc. cit.;* P. 11

21. "Although it is often said that bacteremia accompanying hemolytic streptococcal infections can produce severe anemia due to hemolysis, it is obvious from the lack of signs of rapid blood destruction, such as icterus and hemoglobinura, that this statement is in error. Jaundice occurs in some staphylococcal and Salmonella bacteremias but the bilirubinemia results from toxic hepatitis rather than from any hemolytic action of the causative organisms." I. Bennett; *loc. cit.;* P. 12

Erroneous Idea

22. "In disseminated infections due to *Clostridium welchii,* invasion of the blood stream is characterized by rapid development of icterus, hemoglobinuria, and severe anemia; the development of this clinical picture should suggest the possibility of such an infection." I. Bennett; *loc. cit.* P. 12

23. "Another erroneous idea that is widespread relates to the possibility of producing meningitis by performing lumbar puncture upon a patient with bacteremis." I. Bennett; *loc. cit.* P. 12

24. "In some patients with Laennec's cirrhosis, there occur spontaneous bouts of bacteremia caused by organisms which are normal inhabitants of the intestine, usually the colon bacillus. Although this type of bacterial invasion of the circulation is usually regarded as a rarity, the writer has been impressed with its frequency when it is searched for." I. Bennett; *loc. cit.;* P. 13

25. "Contrary to statements which advocate the immediate administration of antibiotics *intravenously* if bacteremia is present, therapy should always be directed at the primary focus of infection in the tissues. Bacteremia is simply an indication of spreading infection and, as has been detailed above, the blood itself is endowed with enormous powers of defense." I. Bennett; *loc. cit.;* P. 13

Clinical Notes From Grand Rounds

These notes are abstracts of opinions expressed by staff members during case presentations at the Friday Grand Rounds of the Pratt Diagnostic Clinic — New England Center Hospital.

Cobalamin (Vitamin B_{12}) may be made radioactive by using radioactive cobalt (CO^{60}). This substance has been found to be quite useful in the diagnosis of pernicious anemia. In normal individuals, cobalamin is absorbed from the gastrointestinal tract and stored in the liver; when the radioactive material is used, it may be detected by using a Geiger counter over the liver. In pernicious anemia there is no uptake from the gastrointestinal tract, and the counter detects no radioactivity over the liver. In pernicious anemia, radioactive cobalamin appears in high concentration in the feces and in very low concentration in the urine; when intrinsic factor is given, these determinations become normal, and radioactivity may be detected over the liver. There is no defect in absorption of cobalamin in the pernicious anemia of pregnancy. In sprue, following oral administration, there is a high content of cobalamin in the stools, but this cannot be restored to normal by the addition of intrinsic factor.

Truths and Concepts Concerning Orthopaedics

By Morton H. Leonard, M. D., El Paso

This represents a departure from Dr. Babey's conduct of his column in that he has invited guest columns. Other physicians are invited to submit collections of aphorisms on Medical Subjects.

1. Treat shock first.

2. The abdominal distention accompanying fractures of the vertebral body is usually, but not always, a paralytic ileus.

3. A compound fracture is sensational; a ruptured viscus can be lethal.

4. The urethra or bladder can be ruptured in the presence of only a minimal pelvic fracture.

5. The trauma which causes a disruption of bony continuity sometimes causes a disruption of neural and/or vascular continuity.

6. Thrombembolic phenomena are common in fractures in and about the pelvic girdle.

7. A rise in pulse not paralleled by a commensurate rise of temperature may mean shock or a "gas infection."

Gas Bubbles

8. Gas bubbles in the tissue are not always due to "gas gangrene."

9. Pain in the suboccipital region on opening the mouth may indicate upper cervical spine pathology.

10. If the jaw swings to one side as the mouth is opened look for pathology in the temperomandibular joint on the side to which the mandible "points."

11. Negative x-rays do not rule out a significant injury to the neck.

12. A negative x-ray does not rule out metastasis to the skeleton.

13. A negative x-ray does not rule out an early fatigue fracture.

14. Neck pain may have its origin in the shoulder.

15. Shoulder pain may have its origin in the neck.

16. Primary subdeltoid bursitis is unusual.

17. Anti-biotics will not penetrate "dead bone."

Two Weeks Behind

18. X-ray findings in osteomyelitis lag two weeks behind the pathologic anatomy.

19. Dead tissue is an excellent culture medium.

20. Non-specific pus "dissolves" hyaline cartilage.

21. A negative x-ray and a normal sedimentation rate do not rule out an early ankylosing spondylitis.

22. The patient with bone and joint tuberculosis has a systemic illness.

23. The patient with recurrent renal calculi may have hyperparathyroidism.

24. The patient with recurrent renal calculi may have hyperuricuria.

25. In the patient with Vitamin D resistant rickets check the kidneys for abnormality.

Hip Deformities

26. Congenital hip deformities are far more common in the female.

27. Competent treatment of congenital dislocation or subluxation of the hip prior to the age of one year usually results in an excellent hip.

28. Treatment of congenital dislocation of the hip after the age of two years rarely produces an excellent hip.

29. Progression of idiopathic soliosis stops at the time of sexual maturity.

30. To diagnose minimal slipping of femoral capital epiphysis one must have a lateral as well as an AP x-ray.

31. Most children age two to seven years are slightly knock kneed.

32. Most children are "flat footed" to the age of 24 months.

33. If subastragular motion is diminished rule out a congenital fusion in the hind foot.

Easiest Treatment

34. The easiest treatment for a bunion is a wide shoe.

35. A flat foot is not necessarily a painful foot.

36. No cast gives complete immobilization.

37. All wrist sprains are fractures of the carpal scaphoid until proven otherwise.

38. "Edema is [joint] glue," Watson Jones, Reginald: Fractures & Joint Injuries, Vol. 1, Baltimore, 1944, Williams & Wilkins Co.

39. The most common residual disability from Colles' fracture is stiffness of fingers and/or shoulder.

40. Epiphyseal separations of the shearing type only rarely cause growth disturbances; those of the impacting type commonly do.

41. Most "discs" get well on conservative management.

*I am grateful to Dr. Leonard for his contribution. He wishes it understood that these aphorisms are not original with him. He can vouch, on the oasis of personal experience, for their reliability. — Andrew M. Babey, M. D.

The Use of Chemotherapy as an Aid to Hemorrhoidectomy Convalescence

By H. D. Cogswell, M. D., E. W. Czerny, M. D., and W. A. Soland, M. D., Tucson, Arizona

We feel that any means of decreasing the post-operative pain and hastening the convalescence after a hemorrhoidectomy should be brought to most surgeons' attention. This operation has a bad reputation with the general public. It isn't that the operation is unsuccessful or the end result unsatisfactory, but the average post-hemorrhoidectomy patient talks little of his eventual relief of symptoms.

More often he indoctrinates his listening audience in the discomforts and horrors of the "getting well" period. Certain complications such as prolonged healing, post-operative fissures, stenosis, and the varying complaints of the patients have made many surgeons dislike the operation. Because of the self-advertised discomfort of hemorrhoidectomy patients, many individuals needing the operation have avoided operative procedures, either patronizing quacks or suffering needless disability.

Varying Success

The injection of long lasting anesthetics and the application of various ointments and lotions have been recommended to alleviate post-operative pain. These have had only varying success, certainly not with enough constant relief to find them universally adopted.

In observing a number of patients who had very little post-operative pain and a rapid convalescence, we were interested to find that in most of these cases the operated areas were clean without evidence of gross infection. Those with a longer and more painful convalescence showed redness, swelling, and a purulent drainage in and around the rectal wounds.

From this observation, we concluded that if hemorrhoidectomy wounds could be protected from gross infection, the healing period would be more comfortable and shortened. For this reason, a pre-operative regimen was started, using chemotherapy as an intestinal antibacterial agent three to four days before hemorrhoidectomy.

Results Good

The chemotherapeutic agents, usually sulfathaladine or sulfasuxidene, were continued during the healing period. In 1954 (1) the results of our first seventy cases who had been placed on this regimen were reported. At that time the results were good, with a much less painful post-operative period and a shorter healing time. We have continued this procedure and are further convinced that our results are as good as, or better than, we originally reported.

We have followed no specific criteria in measuring the good our patients have received from the pre- and post-operative administration of chemotherapeutic drugs.

More Comfort

It has been observed, however, that in addition to a much greater degree of post-operative comfort and a return to their occupations in a much shorter period of time, these patients require less narcotics and catheterizations, and had fewer post-operative complications. Our method of hemorrhoidectomy and the type of patient have not changed to account for the improvement in our post-operative results.

There have been few instances of drug sensitivity, and when these have occurred we have varied the drug used, antibiotics being substituted. In most operative cases we have found healing to be complete in three weeks and believe that our hemorrhoidectomy patients are healing more comfortably and rapidly because of the addition of chemotherapy to control wound infection.

(1) Cogswell, H. D.; Czerny, E. W.; Fritz, J.: The Use of Chemotherapy as an Adjunct to Healing following Hemorrhoidectomy. Arizona Medicine, Volume 11, No. 5, Page 169. 1954.

Speakers Announced for Meeting of Ob-Gyn Academy in Tucson

Speakers for the annual meeting of District 8 of the American Academy of Obstetrics and Gynecology have been announced by Drs. H. S. Rhu, Jr., and L. D. Sprague, both of Tucson and co-chairmen of the meeting, which will be held April 9-11 in the Pioneer Hotel in Tucson.

Among speakers at the session will be:

David Figg, M.D., Seattle.
Chester L. Roberts, M.D., Glendale, Calif.
Robert Hall, M.D., Salt Lake City.
John Brougher, M.D., Vancouver, Wash.
Don de Carle, M.D., San Francisco.

Approximately 200 doctors and their wives are expected to attend.

Dr. R. Glenn Craig of San Francisco, chairman of District 8 will open the meeting with an address at 9:30 a. m. April 9.

An honored guest will be Dr. Ralph E. Campbell, professor of obstetrics and gynecology at the University of Wisconsin School of Medicine, who is president of the American Academy of Obstetrics and Gynecology.

"Fun in the Sun and Science in the Shade" will be the slogan for the meeting. This slogan was devised by Donald Richardson of Chicago, executive secretary of the American Academy of Obstetrics and Gynecology, who also will attend the meeting.

MONTHLY CLINICAL PATHOLOGICAL CONFERENCE
EL PASO GENERAL HOSPITAL

FREDERICK P. BORNSTEIN, M. D., EDITOR — CASE NO. 542
PRESENTATION OF CASE BY DR. BRANCH CRAIGE

History — Dr. Nathan Kleban

A 31-year-old unmarried Latin-American cardiac clinic patient entered El Paso General Hospital for her 12th admission on September 4, 1955.

The mother's pregnancy and the patient's birth and neo-natal histories were not recorded. She was said to have had measles, whooping cough, a back injury, rickets, and Pott's disease in childhood.

In 1937, at the age of 12, the patient was first seen at an El Paso General clinic, with the notation "no diagnosis." Epistaxis attributed to chronic rhinitis brought her to the clinic in 1939. For this, she was hospitalized in 1941, discharge diagnosis being epistaxis, Pott's disease and rheumatic mitral stenosis. There was no record of cyanosis. No statement was found in the chart as to when cyanosis, which was intermittent, first appeared and as to what events it was related.

Rapid Heart

Not until January, 1949, was she seen again, when she was admitted for complaints of a "bad heart for a long time," ankle swelling, orthopnea, paroxysmal nocturnal dyspnea and rapid heart beat for one month. There were severe kyphoscoliotic deformity of the thoracic spine, pigeon breast, cyanosis of the finger nail beds, spooning as described by one examiner and "chronic pulmonary osteoarthropathy" by another. Rales, liver enlargement and edema were not present. One examiner described the heart as enlarged to left and right, having a loud whistling systolic murmur heard at all valve areas, and a deep rough diastolic murmur loudest over the aortic but also heard over the pulmonic area. Another examiner described the murmur as "machinery like" over the precordium.

Two Diagnoses

The R. B. C. was 5.5 million, Hb. 91 per cent, Kline negative, as it was on all subsequent admissions, zero erythrocyte sedimentation rate, and trace of albumin in the urine. Electrocardiograms: Sinus tachycardia. Cardiac fluoroscopy and chest film: "Marked left ventricular hypertrophy with moderate generalized cardiac enlargement. The aorta was not transposed. The very marked scoliosis of the spine makes it impossible for a definite diagnosis as to type of heart disease, but it is likely that tetralogy of Fallot does not exist." A hilar dance was not observed. The patient was digitalized. Discharge diagnosis was "congenital heart disease with interventricular septal defect."

Because of "palpitation, weakness, turning purple and dizziness", the patient was readmitted in March, 1949. Blood pressure 130/80. Mild cyanosis of the lips and finger nail beds with finger clubbing were described. Deformities of the legs including the pes cavus were noted. There was a high pitched systolic murmur over the entire precordium but no diastolic murmur. A2 was rough and prolonged. Discharge diagnosis was "congenital heart disease with dyanosis tardive."

Fourth Admission

Fourth admission for oxygen therapy to relieve cyanosis occurred one month later. Diagnosis: Congenital heart disease, incompletely diagnosed.

The patient was followed in the cardiac clinic in 1949. She was said to have "hysterical seizures." Because of a persistent heart rate above 100, thiouracil was prescribed for a possible hyperthyroidism, but later discontinued. There were B.M.R.'s of plus 71 and plus 3. On one occasion, the patient was said to be fibrillating and on others premature ventricular systoles were noted. On one clinic visit the examiner heard a systolic murmur loudest in the third left intercostal space and a diastolic murmur, location not specified. Digitalis and phenobarbital were prescribed.

Fifth Admittance

For the next three years the patient was not seen at El Paso General Hospital. In June, 1953, she was admitted for the fifth time, complaining of shortness of breath, abdominal cramps, chest pain, and inconstant ankle swelling. B. P. 110/70; P. 150 regular with premature systoles. Rales were heard. The heart was thought to be enlarged to right and left by one examiner, who described systolic and diastolic murmurs. A consultant thought the right ventricle was enlarged, described a high pitched systolic murmur maximal in the third and fourth intercostal spaces 5 cm. to the left of the sternum.

Liver was not enlarged, edema not present. Hb. 16.2 grams, R.B.C. 5.2 million, W.B.C. 6900, blood urea nitrogen 11 mg. per cent. Urine 1,025, negative. Chest films: "Kyphoscoliosis with convexity to right in upper portion. No cardiac enlargement. Intensification of pulmonary markings. Findings consistent with congestive failure, old acid fast spondylitis and kyphoscoliosis". The patient was digitalized, and six days later a repeat chest film was interpreted as demonstrating improvement of the congestive failure.

Diagnoses

The P waves in leads one and two were slurred on electrocardiogram. Diagnoses were "Kyphoscoliotic pulmonary heart disease and possible congenital heart disease".

Admission in December, 1953, was for "heart pain and dizziness". Hb. 16.2 grams; blood urea nitrogen 22.5 mg. per cent; P. S. P. excretion 43 per cent and 60 per cent in 2 hours; trace of albumin and hyaline and granular cysts in the urine. The patient was sedated and given mercurial diuretic.

Wheezes and Rales

During 1954, weight varied between 74 and 78 pounds. Blood pressure varied between 160/130 to 120/90. Wheezes and a few rales were heard. A grade IV systolic murmur over the entire precordium and a diastolic murmur at the apex, were described. The liver was not felt and there was no edema. Hb. 16.7 grms.; urine 2 plus albumin, 1,024; B. U. N. 10 mg. per cent, Demerol, Raudixin and an antibiotic were administered. In October and November, the patient was admitted with complaints of "heart attacks." A chest film was reported as demonstrating "no significant change." The usual diagnosis was "Kyphoscoliostic heart disease". On one admission, there was a second final diagnosis of "hypertension".

On clinic visits during 1955, weight was 79-80 pounds, blood pressure not recorded, and no evidence of congestive heart failure noted. Medications were: Thorazine and Raudixin.

Tenth Admission

Tenth admission was in January, 1955. Blood pressure was 100/80, systolic and diastolic murmurs were heard over the entire chest; heart size was indeterminate because of chest deformity; liver was not felt; and there was no edema. Urine: one plus albumin and granular casts. The house staff complained of the patient's demand for enemas and refusal to expel them.

In June, 1955, the patient was admitted because of menorrhagia. Pelvic examination was not remarkable. Bleeding ceased after estrogen was given. A systolic murmur loudest at the base and a diastolic murmur loudest at the apex were described. Blood pressure was 120/80. Other medications were penicillin, digitalis and sedatives. Diagnoses were Kyphoscoliotic and rheumatic heart disease with mitral stenosis.

The patient entered the hospital on September 9, 1955, for the twelfth and final admission for reasons not found in the record.

There was neither family nor past history of rheumatic fever.

Physical Examination

T. 99.4, P. 110, B. P. 130/90, wt. 80 lbs. State of nutrition was poor, skin pale, lips and finger nail beds were cyanotic. One examiner heard rales at the lung bases, another did not. There were premature systoles. Liver was not felt. There was no edema.

Hospital Course

The patient appeared to be in no respiratory distress during most of her hospital stay. She complained of headaches, chest pains, abdominal cramps, nausea, vomiting, and inability to sleep. Temperature was not elevated except on two occasions, when it was 100 and 101 and rose to 104 on the day of death.

She demanded medications and injections, cried for her mother, screamed at night, and was transferred to the Annex when she was disturbed by vision of a legless man and threatened to jump out the window.

On the 20th day in hospital, two plus ankle edema was noted. Generally the patient ate in the dining room, watched TV, went to the phone to call her mother, and demonstrated no striking changes until the 35th hospital day.

She was found to be dyspneic, cyanotic, without detectable blood pressure. There was periorbital but not pedal or other edema described. Oxygen, aminophylline, Levophed, and I-V Cedilanid were administered.

Consultant's Note

A consultant's note stated: "Patient was terminal without blood pressure and a tachycardis of 160, occasional PVC, Pulsus alternans 140-120. Prognosis poor".

Blood pressure rose to 140/100. On the 36th day there was cyanosis if oxygen was stopped, agonal breathing, bubbling rales, and bloody emesis. Blood pressure was 110/160 with levophed. Bowel sounds were absent.

There was abdominal distention, coffee-ground vomitus. Wangensteen suction was instituted. The patient died in an oxygen tent on the 37th day.

Additional medications given were: Phenobarbital, A.P.C., Chloral hydrate, Thorazine, Nembutal, Seconal, Demerol, Raudixin, Dormison, Memphate, Benadryl, Serpasil, Moludar, Diamox, Penicillin, Mercuhydrin, and Achromycin.

Laboratory

X-Ray Oct. 10, 1955: Chest film, portable. "Chest not diagnostic due to the condition of patient."

Lab. Sept. 4, 1955: Hb. 14.2 grms.; RBC 4.36; WBC 9150; Stabs 2, Segs 81, lymphs 13; monos 4.

Urinalysis: Yellow, acid, 1,005, albumin 2 plus, Sugar Negative, WBC 3-5/HPF

KLINE Exclusion negative

Erythrocyte Sedimentation rate 4 M M/hr.

EKG Sept. 28, 1955: Sinus tachycardia with frequent ventricular premature systoles. Tall peaked P waves in Lead II. Horizontal heart, slight left axis, T waves low. "ABNORMAL record suggesting myocardial damage."
Oct. 9, 1955: Sinus tachycardia, PVC's, P tall and peaked in II.

X-Ray Discussion — Dr. Vincent M. Ravel

Three or four examinations of the patient show a tuberculous spondylitis with a marked Kyphoscoliosis. The problem of evaluating a cardiac silhouette with as much deformity as this is considerable. On one occasion, it was stated that the heart shadow was enlarged, and on another occasion it wasn't. I do not believe there is any significant cardiac enlargement.

I think what has happened is that the deformity of the chest makes for a greater ratio between the cardiac silhouette and the general space within the chest cavity. It makes it look like a relative cardiac enlargement; yet, actually I do not think that the heart shadow is significantly enlarged. I think it is a matter of having less chest space.

Difficult Problem

The problem of congenital heart disease is, I think, rather difficult to evaluate, but I do not believe again that this is a congenital heart. Circulation depends, of course, to a certain extent upon the ability of the diaphragm to produce a satisfactory negative pressure in the chest which, in turn, causes a considerable assistance so far as return blood flow to the heart is concerned.

Here we have a problem of limitation of motion of the diaphragm with an additional hemodynamic strain. The changes that we see definitely are the tuberculous spondylitis, and I believe that there will be considerable right heart enlargement as compared with the left ventricle.

Final Film

The final film that we see was taken when the patient was in extremis and I am sure that there must have been evidence of considerable congestive failure, filling up of the left chest cavity with a pleural effusion. The problem of a tuberculour pericarditis with constriction must be considered in view of the original lesion of the tuberculous spondylitis.

Differential Diagnosis — Dr. Branch Craige

I want to start out by thanking Dr. Kleban for a good summary of a very long record. I don't intend to thank Dr. Bornstein for picking this particular case, though.

This women had ten admissions to the hospital, during which time observations were recorded, I presume, by a score or more of observers, many of which Dr. Kleban has incorporated here so that we have observations pro and con for almost any diagnosis we would like to choose.

The problem, of course, the big problem, is what kind of heart disease, if any, did she have? On various admissions to the hospital, she had diagnoses made of Kyphoscoliotic heart disease, mitral stenosis, and of congenital heart disease, the type of which was not specified.

Other Diagnoses

Let's leave the discussion of the heart disease to the last, and dispose first of a number of various other diagnoses and possible diagnoses which are suggested by the record. The diagnosis of Pott's disease was fine. I think we should accept that. She is said to have had a back injury and rickets in childhood. We are unable to corroborate those. We will accept those at their face value.

At one place in the records it is mentioned that she had pes cavus deformities of the feet, which raises a question of whether instead of a tuberculous spondylitis, she might have had one of the muscular dystrophies with damage to the feet and the chest at the same time. I don't think the information available is enough to do any more than speculate on that score. I think we will have to accept the diagnosis of Pott's disease. However, chest deformities like this can occur in some of the myopathies.

Epistaxis Mentioned

Epistaxis is mentioned, and that, of course, raises the suggestion—epistaxis, that is, in childhood—of rheumatic fever, of rheumatic heart disease. A renal lesion is suggested by the appearance of albumin and casts in the urine at one time, an elevated B. U. N.—slightly elevated—22—but later it went down to 11. Hypertension is noted in one spot and subsequent blood pressures were recorded as normal. However, it didn't specify whether the normal blood pressures were recorded when she was under the influence of Raudixin. Can you tell me?

Answer — Blood pressures were normal with and without Raudixin.

Dr. Craige: Blood pressures were normal with and without Raudixin. I would be inclined to discount the importance of the isolated hypertensive readings. She had menorrhagia at one time, relieved by estrogens, which I would interpret in the absence of any other findings as not significant. She was thought at one time to have hyperthyroidism, the only evidence for which is a single BMR plus 71, and a nervous disposition. I think that we do not have enough evidence to corroborate that diagnosis.

Psychotic Symptoms

In her last admission, she developed psychotic symptoms, but that was at a time when she was pretty sick, and I would suggest that the psychotic symptoms were probably due to a sympotmatic psychosis.

Now, we come to "What kind of heart disease does this woman have?".

First, with regard to rheumatic heart disease, the one murmur that everybody agreed on was that she had a loud systolic murmur all over the precordium. A diastolic murmur was recorded on some occasions at or near the apex—or wherever the apex might have been thought to be—and with the history that I have mentioned previously, rheumatic heart disease and mitral stenosis were mentioned.

Left Axis Deviation

However, this girl had left axis deviation on the electrocardiogram. Going by the records, at least, on fluoroscopy the left ventricle was thought to be hypertrophied. There was no significant evidence of ventricular enlargement and my feeling would be that she did not have rheumatic heart disease.

Congenital heart disease was diagnosed on some occasions. That would have to be either cyanotic or not cyanotic or potentially cyanotic. Well, now, with regard to the cyanotic congenital heart diseases, this girl did not have any right ventricular hypertrophy. There was no right axis deviation in the cardiogram. She was not constantly cyanotic. I think there is no evidence of her having a cyanotic congenital lesion.

Patent Ductus

Of the non-cyanotic lesions, I need not go into them because I think there is little evidence for them, except for the question of a patent ductus. One of the doctors thought at one time she had a "machinery-like murmur" over the base of the heart. Patent ductus, however, shouldn't make her cyanotic.

Of the potentially cyanotic lesions, septal defects and perhaps patent ductus and pulmonic stenosis should be thought of. This girl apparently developed cyanosis from time to time without any marked respiratory distress at the time that the cyanosis ocurred. I would be inclined to think that she did not have any congenital heart disease.

Kyphoscoliotic Heart

Now that leaves us with kyphoscoliotic heart disease, and I don't see any reason why all of the signs and symptoms which this girl presented could not fit into that diagnosis. She did have, obviously, a severely deformed chest. The cardio-thoracic ratio is altered, but I think it is the thoracic end of the ratio that is altered rather than the cardiac.

Shrunken Chest

I think she has a normal sized heart and a shrunken chest. The left axis deviation would make one suspect that maybe this diagnosis is wrong, but in severe Kyphoscoliosis you can get right or left axis deviation. You can get left ventricular hypertrophy and/or right ventricular hypertrophy. She had cyanosis from time to time but cyanosis could occur due to the fact that segments of the lung remained atelectatic and I presume that the blood that flows through those atelectatic areas is fully oxygenated.

She did have club fingers, or at least she had some abnormality of the fingers. Kyphoscoliotic patients often have bronchiectasis. We don't have definite evidence for it in this case but she may well have had it and I don't think that the club fingers would make for any doubt as to the diagnosis.

With right scoliosis as this girl had, the heart is displaced to the left and left axis deviation of the cardiogram is not inappropriate.

Biggest Problem

Now, with regard to the murmurs. The biggest problem, apparently, and the biggest reason for suspecting some other kind of heart disease in this girl, was the fact that she had variously described diastolic murmurs. In Kyphoscoliotic heart disease, the auscultation of the heart is interesting, but useless so far as the differential diagnosis goes, because murmurs may be systolic or they may be diastolic.

I believe they may be heard at any position in the chest and they have lost their significance so far as determining vascular lesions. So, I think that in this 31-year-old girl the choice would be to make as few diagnoses as possible.

I think she has Kyphoscoliosis and I think she has Kyphoscoliotic heart disease, so-called, which is really not disease of the heart at all, but an inadequate chest area, and her terminal event, namely, going into rather rapid and profound and very abrupt ending is characteristic for patients with Kyphoscoliosis, because once failure occurs in such patients, the progress is extremely rapid.

Dr. Appel:

One of the things that wasn't emphasized in the protocol was the — well, I'm no psychiatrist, but she had a severe, I would say, psychosis, associated with this condition. That is one of the reasons why she received Raudixin and Thorazine and barbiturates. In fact, I'm pretty sure she was addicted to some of those drugs, from the remarks Dr. Logsdon made.

I only examined her on the last night of her illness and at that time she was terminal in the Annex and we even got an EKG to see if it would help us, but it didn't make much difference.

Clinical Diagnosis:

Kyphoscoliotic heart disease?
Rheumatic Heart disease?

Dr. Branch Craige's Diagnosis:

Kyphoscoliotic heart disease.

Pathological Diagnosis:

Kyphoscoliotic heart disease. Patent inter ventricular septum, membranous portion.

Pathological Discussion — Dr. Bornstein

The autopsy was performed by Dr. Rita Don, and I am following the protocol. The external examina-

tion showed a small kyphoscoliotic woman, extremely cyanotic, with club fingers. Each pleural cavity contained 100 ccs. of fluid. The pericardial sac contained 500 ccs. of fluid. The heart weighed 480 grams. The right ventricle measured 8, the left ventricle measured 15 mm. in thickness. This represents hypertrophy of both ventricles. In addition, there was a large defect in the interventricular septum which was located in the membranous portion. (Fig. 1.)

Lungs and Liver

The lungs weighed 300 grams each. The liver weighed 940 grams. The remaining organs were not remarkable.

In the interpretation of these findings, it is easy to overvalue the importance of the septal defect for the development of the clinical picture. I think this woman died of Kyphoscoliotic heart disease for the following reasons:

Probable Consequences

What are the probable consequences of a septal defect in this particular case? To me, it means that the left ventricle in contracting throws some arterialized blood back into the pulmonary circulation. This is a mechanism similar to mitral insufficiency. Yet, this woman did not die the death of a mitral insufficiency.

Figure 1

There was no cardiac dilatation. Especially, there was no brown induration of the lungs. I think the septal defect is a contributory cause of death and offers an explanation for the very confusing cardiac murmurs that were heard during the course of the disease.

Cause of Death

Death here is due to Kyphoscoliotic heart disease. In those cases of septal defect where death occurs as a consequence of the defect, the disease usually becomes complicated by a super imposed infection with the development of an endocarditis lenta. They do not die of decompensation.

In summary, then, this was a patient who died after a long chronic Kyphoscoliotic heart disease with symptoms which were confusing due to the presence of a septal ventricular defect.

Dr. Blanchard Elected President of San Miguel County Medical Society

Las Vegas, N. M. Dr. J. J. Johnson, Jr., has returned from the University of Colorado at Denver, where he attended one week of intensive study in general medicine and surgery.

Dr. Junius A. Evans and his wife have returned from Mazatlan, Mexico after a month's stay. On December 7, he had an attack of coronary thrombosis and his physicians advised a three months rest from his busy practice.

Dr. Volney S. Cheney has recently resigned as secretary and treasurer of the San Miguel County Medical Society after serving many years in that capacity. Dr. Cheney is 81 years young and is still very active in public school practice.

Dr. Charles L. Blanchard was recently elected President of the San Miguel County Medical Society. Dr. Blanchard is also the President of the Shrine Club of Las Vegas and Chairman of the Under-privileged Children's Committee of the local Kiwanis Club.

General Practitioner Training In Proctology

Post-graduate courses are listed annually in the J.A.M.A. and in the American Journal of Proctology. Regular reading of the American Journal of Proctology will bring the basic literature on both ano-rectal and colon proctology to the physician.

The International Academy of Proctology offers annual teaching seminars, and quarterly state chapter meetings, all without registration fee. The general practitioner is cordially invited to attend these meetings, and to take an active part in the discussions. The Eighth Annual Teaching Seminar of the International Academy of Proctology will be held in Chicago, April 23 - 26th, 1956, at the Drake Hotel.

MEETINGS

New Mexico Medical Society Announces Speakers for Annual Meeting in Roswell

The seventy-fourth annual session of the New Mexico Medical Society will be held in fast growing Roswell, metropolis of Southeast New Mexico, May 2, 3, and 4.

Guest speakers for the session will be:

Carleton B. Chapman, M. D., Internist, Dallas.

John H. Moyer, M. D., Internist, Houston.

Ian MacDonald, M. D., Surgeon, Los Angeles.

Morris J. Fogelman, M. D., Surgeon, Dallas.

Conrad G. Collins, M. D., Obstetrician and Gynecologist, New Orleans.

Forrest H. Adams, M. D., Pediatrician, Los Angeles.

Dana M. Street, M. D., Orthopedics, Memphis.

Robert D. Moreton, M. D., Radiologist, Fort Worth.

Vernon E. Martens, Capt., MC USN, Maryland.

In addition to the scientific papers there will be two panel discussions. Dr. Collins will moderate the panel on "Acute Anuria" and Dr. Moyer will moderate the panel on "Steriods". All guest speakers will participate on the panels. There will be medical and surgical round-table luncheons on Thursday and Friday.

Friday evening, May 4, has been reserved for meetings of the various specialty groups.

Business Meetings

Business meetings, Council, and House of Delegates of the State Society will be held on Tuesday, May 1, and Wednesday morning, May 2. Details of this phase of the Session will be announced later.

In addition to the scientific program the Chaves County Medical Society, convention host, has arranged a round of social activities, which should help each doctor to relax. Wednesday evening, May 2, has been set aside for the smoker and Thursday, May 3, will be the annual dinner-dance.

Those who care to do some sight-seeing will find the charm of the West in Roswell, which is surrounded by thousands of fertile acres of growing crops and is in the midst of one of the best sheep and cattle range sections of the Southwest.

Carlsbad Caverns

Chief attraction of the Roswell area is world famous Carlsbad Caverns National Park, located 106 miles to the south. To the east is Bottomless Lakes State Park, a unique phenomenon of both scenic and sport interest, where boating, swimming, horse-back riding and picnicking are enjoyed.

To the west, 72 miles, is the famous mountain playground, Ruidoso. The Nation's highest golf course, 9,000 feet, at Cloudcroft, is only 130 miles Southwest of Roswell. Thus, convention participants will have a wonderful opportunity while visiting Roswell to visit some of the most scenic areas of the Southwest.

All physicians who are members of their respective state medical societies are cordially invited to attend.

Room accommodations are available in the Nickson Hotel or one of the many beautiful motels. A list of the motels will be provided in the near future, or write: New Mexico Medical Society, 223-24 First National Bank, Albuquerque.

Dr. Hossley of Deming New President of Southwestern New Mexico Society

Dr. W. J. Hossley, Jr., of Deming, N. M., was elected President of the Southwestern New Mexico Medical Society at its annual meeting in Deming, January 18th, 1956. Other new officers are Dr. C. C. Cobb of Silver City, vice president; and Dr. Sidney F. Baker of Silver City, secretary-treasurer. Dr. J. C. Sedgwick, Las Cruces, N. M., was the retiring president.

Dr. Saul Appel of El Paso presented a paper on "Rheumatic Fever in Pregnancy and Old Age." Also present at the meeting were Dr. Earl Malone of Roswell, N. M., president of the New Mexico Medical Society, and Ralph Marshall, Albuquerque, executive secretary of the New Mexico Medical Society.

The Society voted to hold future meetings in Deming because of its availability to all the component societies. Meetings will be held every four months instead of three as previously, and Deming will be the host Society.